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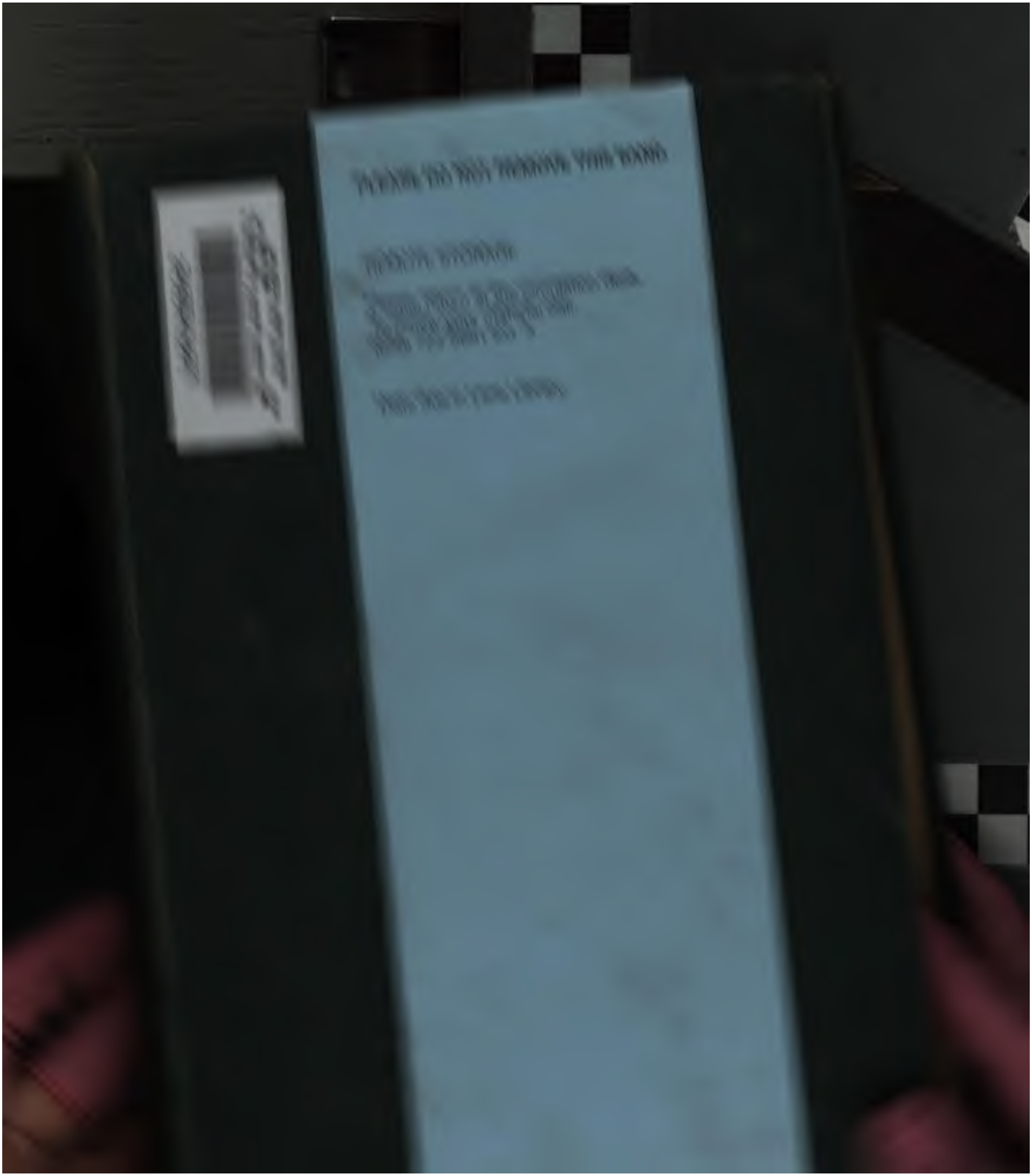

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July 27th 18

H. Cochrane.
PHYSICIAN & SURGEON.
[illegible] SAN JOSE [illegible]

# A TEXT-BOOK

OF

# PRACTICAL MEDICINE,

WITH PARTICULAR REFERENCE TO

# PHYSIOLOGY AND PATHOLOGICAL ANATOMY.

BY

DR. FELIX VON NIEMEYER,
PROFESSOR OF PATHOLOGY AND THERAPEUTICS, DIRECTOR OF THE MEDICAL CLINIC OF THE UNIVERSITY OF TÜBINGEN.

*TRANSLATED FROM THE SEVENTH GERMAN EDITION, BY SPECIAL PERMISSION OF THE AUTHOR,*

BY

GEORGE H. HUMPHREYS, M. D.,
ONE OF THE PHYSICIANS TO THE BUREAU OF MEDICAL AND SURGICAL RELIEF AT BELLEVUE HOSPITAL FOR THE OUT-DOOR POOR, FELLOW OF THE NEW-YORK ACADEMY OF MEDICINE, ETC.,

AND

CHARLES E. HACKLEY, M. D.,
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VOLUME I.

NEW YORK:
D. APPLETON AND COMPANY,
90, 92 & 94 GRAND STREET.
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Entered, according to Act of Congress, in the year 1869, by
D. APPLETON & CO.,
In the Clerk's Office of the District Court of the United States for the
Southern District of New York.

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# TRANSLATORS' PREFACE.

The pleasure and profit derived from the perusal of Dr. Niemeyer's Hand-book of Special Pathology and Therapeutics have led the translators to suppose that an English edition of the work might be acceptable to the medical profession in America.

It is true that, from the present multiplicity of excellent English treatises upon the practice of medicine, such an undertaking may seem superfluous; but the translators cannot but hope that the work in question may, in some degree, supply a want which many of the more recent hand-books do not altogether satisfy. The sciences of Pathology and Therapeutics have made vast strides within the last ten years; and, for very many important researches and discoveries in both these branches of medicine, we are indebted to Germany. Professor Niemeyer's volumes present a concise and well-digested epitome of the results of ten years of carefully-recorded clinical observation by the most illustrious medical authorities of Europe, together with many valuable and practical deductions regarding the causes of disease and the application of remedies, such as we believe have not as yet been assembled in any single work.

The rapidity with which it has passed through seven Ger-

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man editions, the last two of triple size, and the fact that it has already been translated into most of the principal languages of the Old Continent, afford ample proof of its appreciation in Europe, and encourage the translators to hope that, in presenting it in an English form, their labors may not prove wholly useless to their American professional brethren.

NEW YORK, *May* 20, 1869.

# PREFACE TO THE SEVENTH EDITION.

NEARLY ten years have elapsed since the first appearance of my text-book. Meanwhile, clinical medicine owes a rich accession of knowledge to investigations made, not only in her own province, but in the provinces of physiology, pathological anatomy, and physiological and pathological chemistry. Important questions have been settled; obscure points rendered clear; false theories corrected, and errors recognized.

Although, in preparing previous editions, I have taken pains to keep my book well up to the existing state of science, to render account of the most important advances made in the study of medicine and its kindred branches, yet the briefness of the time allowed for this purpose—owing to the rapidity with which editions have been renewed—and the conviction that the true value of many discoveries of supposed importance could be ascertained only by a longer probation, have hitherto deterred me from a full and thorough revision of the entire work. At last, ten years after its first appearance, the proper moment seems to have arrived. The somewhat longer respite now allowed me for my task, is due to the foresight of my publisher, who has made the previous edition of triple the usual size.

In the present (seventh edition), but few portions of the work remain unaltered; and even those few have nearly all undergone revision on previous occasions. Most parts of it have received valuable emendations, and have been enriched by copious additions. I have everywhere paid particular attention to the important results obtained in the domain of therapeutics by recent investigations, partly because I wish my book to maintain the honorable confidence which it has won for itself among practical physicians; partly because I regard the happy progress which therapeusis has made, as the most important acquisition of the last ten years.

This progress I attribute mainly to the fact that, of late years, medical explorers have recognized the only path by which therapeutic science can be advanced, and have followed it with brilliant results. My outspoken assertions of ten years ago have come true. I then denounced the error of postponing all medical treatment of disease, until our knowledge of the action of medicines, and our insight into pathological processes, should be so far advanced, that means of cure would be self-evident. I pronounced this ideal goal to be unattainable, and declared it idle to hope for a time when a medical prescription should be the simple resultant of a computation of known quantities. I lamented that physicians, instead of striving to promote the healing art by their own efforts, should seek aid from the institutes of physiology and pathology, or from the laboratory of the chemist, obtaining now and then an ingenious suggestion, but never gaining an idea serviceable in the relief of an afflicted fellow-creature. I further showed that experiments made with medicaments upon the lower animals, or upon healthy human beings, with all their scientific value, had as yet been of no direct service to our means of treating disease, and that a continuation of such experiments gave no prospect of such service. I finally declared, without reservation, that even the dazzling progress which pathology had made, had been of but little use to therapeutics; that, in spite of new discoveries, our present success at the bedside is scarcely more favorable than that of fifty years ago; nor in the future would pathological investigation promote therapeutic success, unless directed more in accordance with the requirements of general medicine, than has been done hitherto.

Thus, after showing that therapeusis must expect no aid from other incomplete sciences, and that it must be conducted by itself as an independent and peculiar branch of knowledge; after showing, further, that the empirical method of investigation is the only rational and proper one for the study either of therapeutics, or of any other department of natural science, I pointed out more precisely what material we already possessed for the establishment of therapeusis as an independent empirical study; showed what still remained to be done, and how that which is still lacking is to be obtained. I then demonstrated that, before all else, empirical knowledge requires a profound and thorough acquaintance with facts, and that the more accurate the observation, so much the more correct and trustworthy must the deduction be, and that observations in therapeusis, if inaccurate or imperfect, are prolific of false conclusions and of erroneous proceedings, just as in other branches of natural science. I explained that, when ancient therapeutic laws, based sometimes upon the experience of centuries, have proved false, the error has been due to inexact and incomplete observation; that the general impression that a remedy has done good or harm, in this or that disease, is utterly worthless in a scientific point of view. I declared that empirical matter, capable of affording trustworthy and useful rules for the treatment of disease, is only to be obtained by the most careful and intelligent investigation of the healing effects of medicaments; that no sure basis for therapeusis can be established until this shall occur; until clinical teachers and physicians, particularly those at the head of the science, familiar with all the accessories to diagnosis, shall comprehend that their main task is, most carefully (and, where possible, objectively) to analyze the symptoms of a disease, prior to and subsequent to the administration of a supposed remedy; that such (no doubt very laborious) investigations have hitherto been totally neglected, because no one expected to obtain any results from such a method of study; but that these pessimist views evince an undervaluation of the brilliant progress of physical diagnosis, of physiology, of pathological anatomy, and pathological chemistry, made in the last ten years. Although direct and immediate advantage to the art of healing is not to be expected of any of these branches of learning, yet every new discovery, in its way, tends to benefit that art, either by improving our knowledge of disease, or by assisting our comprehension of the *modus operandi* of medicines. My conviction is, that from the present state of knowledge, from our deeper insight into the origin and relation of symptoms from the improved accessories, by means of which we are now enabled to follow the various phases and modifications of disease, the prospect of obtaining sure and authentic therapeutic facts, by dint of accurate comparison of results, is not only by no means unfavorable, but, judging from present experience, is positively certain.

Seven years ago, I closed my inaugural address at Tübingen with the following words: "The task is a laborious one; the "difficulties are great; but the knowledge that this is the sole "path leading to the wished-for goal, the conviction that the "smallest well-authenticated fact in therapeutics is of profound "importance, will inspire the perseverance in research requisite "to make therapeusis an exact science, a science which may take "equal rank with other branches of physical study." I may now say that my anticipations have been well-nigh surpassed. A band of distinguished teachers have carried out these laborious researches with a thoroughness and perseverance which could not fail in its effect. The valuable labors, now under prosecution, in the long-neglected field of treatment of disease, by means of which, already, the value of certain important articles, hitherto ill-appreciated, has been accurately determined, have received general recognition, and thus a final blow has been given to the dominion of a disheartening therapeutic nihilism. This success, as an example of which I will merely mention the discovery of the antipyretic action of quinia in typhus, pneumonia, etc., and the establishment of precise indications for the use of digitalis in disease of the heart, has caused the zeal for therapeu-

H. G. Cochrane.
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tic experimentation to assume a direction destined to lead to great results. Rightly supposing that even the rude experience of the ignorant laity and their belief in the all-healing power of the "cold-water cure" and the "bread cure" have some foundation in fact, the effects both of hydropathic treatment and that of the continued limitation of the supply of water to the system have been subjected to rigid analysis. Such laudable abnegation of sectarian pride has been richly rewarded. Among other results, we owe to it our more accurate knowledge of the effect produced by active abstraction of heat upon the temperature of the body in acute febrile disease. This alone is a great achievement. By its means, a weapon, powerful for good in time of peril, is taken from the hands of the laity, where it has done much harm, and, under control of educated and experienced men, who know its capacity and how to regulate its effect, it has become the common property of science. It is a favorable sign that the warm recognition and support formerly enjoyed by the so-called "doctrine of Rademacher"—that wonderful offspring of a clear perception of the errors and failings of traditional therapeutic rules, and of blind submission to the obsolete teachings of Paracelsus—have become extinct; and still better, that the number of pure and devout homœopaths, who, implicitly trusting in homœopathic tenets and doses, make no use of the developments of therapeutic research, has grown small.

May these words, and the contents of my book, aid clinical investigation in pursuing more and more the path by which alone its immediate and main object—the establishment of therapeutic facts—is to be attained!

In conclusion, I tender my cordial thanks to my numerous friends and patrons for their favor; especially to my honored friend and colleague Professor Seitz, of Giessen, for the good counsel with which he has assisted me in the preparation of my new edition. (Signed)

FELIX VON NIEMEYER.

TÜBINGEN, *October*, 1867.

# TABLE OF CONTENTS OF VOL. I.

## DISEASES OF THE RESPIRATORY ORGANS.

### SECTION I.

### *AFFECTIONS OF THE LARYNX.*

PAGE

CHAP. I.—Hyperæmia and Catarrh of the Larynx, . . . . . . . . . 1
II.—Croup, Angina Membranacea, . . . . . . . . . . 15
III.—Catarrhal Ulcers of the Larynx, . . . . . . . . . . 31
IV.—Typhous and Variolous Ulcers of the Larynx, . . . . . . . 33
V.—Syphilitic Diseases of the Larynx, . . . . . . . . . . 35
VI.—Tubercular Ulceration of the Larynx, . . . . . . . . . 38
VII.—Growths in the Larynx, . . . . . . . . . . . . 43
VIII.—Œdema Glottidis, . . . . . . . . . . . . . 45
IX.—Laryngeal Perichondritis, . . . . . . . . . . . 49

NERVOUS DISEASES OF THE LARYNX.

X.—Spasmus Glottidis, . . . . . . . . . . . . . 51
XI.—Paralysis of the Muscles of the Glottis, . . . . . . . . 54

### SECTION II.

### *DISEASES OF THE TRACHEA AND BRONCHI.*

CHAP. I.—Hyperæmia and Catarrh of the Air-passages, . . . . . . . 59
II.—Croupous Inflammation of the Trachea and Bronchi, . . . . . 84
III.—Spasm of the Bronchi, Bronchial Asthma, . . . . . . . 86
IV.—Spasm of the Respiratory Muscles, . . . . . . . . . 91
V.—Whooping-cough, Tussis Convulsiva, . . . . . . . . 92

### SECTION III.

### *DISEASES OF THE PARENCHYMA OF THE LUNGS.*

CHAP. I.—Hypertrophy of the Lung, . . . . . . . . . . . 104
II.—Atrophy of the Lung, Emphysema Senile, . . . . . . . . 105
III.—Emphysema of the Lung, . . . . . . . . . . . . 106
IV.—Collapse of the Lung, . . . . . . . . . . . . . 126
V.—Hyperæmia, Œdema of the Lung, . . . . . . . . . . 130
VI.—Bronchial Hæmorrhage, . . . . . . . . . . . 141
VII.—Pulmonary Hæmorrhage, . . . . . . . . . . . . 153
VIII.—Apoplexy of the Lung, . . . . . . . . . . . . 161
IX.—Croupous Pneumonia, . . . . . . . . . . . . 162
X.—Catarrhal Pneumonia, . . . . . . . . . . . . 190
XI.—Induration of the Lung, . . . . . . . . . . . . 195
XII.—Gangrene of the Lung, . . . . . . . . . . . . 203

TUBERCULOSIS OF THE LUNG.

PAGE
CHAP. XIII.—Pulmonary Consumption, . . . . . . . . . . . 208
XIV.—Acute Miliary Tuberculosis, . . . . . . . . . . 247
XV.—Cancer of the Lung, . . . . . . . . . . . . 251

## SECTION IV.

### *DISEASES OF THE PLEURA.*

CHAP. I.—Inflammation of the Pleura, . . . . . . . . . . 253
II.—Hydrothorax, . . . . . . . . . . . . . . 273
III.—Pneumothorax, . . . . . . . . . . . . . . 276
IV.—Tuberculosis of the Pleura, . . . . . . . . . . 283
V.—Cancer of the Pleura, . . . . . . . . . . . . 284

---

## APPENDIX TO THE DISEASES OF THE RESPIRATORY ORGANS.

### *DISEASES OF THE NASAL CAVITIES.*

CHAP. I.—Catarrh of the Nasal Mucous Membrane, . . . . . . . 286
II.—Bleeding at the Nose, Epistaxis. . . . . . . . . . 292

---

## DISEASES OF THE CIRCULATORY ORGANS.

## SECTION I.

### *DISEASES OF THE HEART.*

CHAP. I.—Hypertrophy of the Heart, . . . . . . . . . . 297
II.—Dilatation of the Heart, . . . . . . . . . . . 316
III.—Atrophy of the Heart, . . . . . . . . . . . . 325
IV.—Endocarditis, . . . . . . . . . . . . . . . 328
V.—Myocarditis, . . . . . . . . . . . . . . . . 340

VALVULAR DISEASES OF THE HEART.

VI.—Insufficience of Semilunar Valve, . . . . . . . . . 345
VII.—Insufficience of Mitral Valve, . . . . . . . . . . 351
VIII.—Insufficience of Semilunar Valve and Contraction of the Mouth of the Pulmonary Artery, . . . . . . . . . . . . . 359
IX.—Insufficience of the Tricuspid, . . . . . . . . . . 360
X.—Degeneration of the Substance of the Heart, . . . . . . . 361
XI.—Rupture of the Heart, . . . . . . . . . . . . 364
XII.—Fibrinous Deposits in the Heart, . . . . . . . . . 365
XIII.—Congenital Anomalies of the Heart, . . . . . . . . 366
XIV.—Neuroses of the Heart, . . . . . . . . . . . 370
XV.—Basedow's Disease, . . . . . . . . . . . . . 374

## SECTION II.

### *DISEASES OF THE PERICARDIUM.*

CHAP. I.—Pericarditis, . . . . . . . . . . . . . . . 378
II.—Adhesion of the Heart and Pericardium, . . . . . . . . 389
III.—Hydropericardium, . . . . . . . . . . . . . 391

PAGE
CHAP. IV.—Pneumopericardium, . . . . . . . . . . . . . 393
V.—Tuberculosis of the Pericardium, . . . . . . . . . . 395
VI.—Cancer of the Pericardium, . . . . . . . . . . . 395

### SECTION III.

#### *DISEASES OF THE GREAT VESSELS.*

CHAP. I.—Inflammation of the Coats of the Aorta, . . . . . . . . . 396
II.—Aneurism of the Aorta, . . . . . . . . . . . . . 400
III.—Rupture of the Aorta, . . . . . . . . . . . . . . 409
IV.—Stricture and Obliteration of the Aorta, . . . . . . . . 410
V.—Diseases of the Pulmonary Artery, . . . . . . . . . . 411
VI.—Diseases of the Great Venous Trunks, . . . . . . . . . 412

---

## DISEASES OF THE ORGANS OF DIGESTION.

### SECTION I.

#### *DISEASES OF THE MOUTH.*

CHAP. I.—Catarrh of the Mouth, . . . . . . . . . . . . . 414
II.—Croupous Stomatitis, Aphthæ, . . . . . . . . . . . 420
III.—Diphtheritic Stomatitis, Cancrum Oris, . . . . . . . . . 422
IV.—Excoriations and Ulcers of the Mouth, . . . . . . . . . 424
V.—Syphilitic Affections of the Mouth, . . . . . . . . . . 427
VI.—Scorbutic Affections of the Mouth, . . . . . . . . . . 428
VII.—Soor, Muguet, Thrush, . . . . . . . . . . . . . 430
VIII.—Glossitis, . . . . . . . . . . . . . . . . . 432
IX.—Gangrenous Sore Mouth, . . . . . . . . . . . . . 434
X.—Parotitis, Mumps, . . . . . . . . . . . . . . . 436
XI.—Salivation, Ptyalism, . . . . . . . . . . . . . . 441

### SECTION II.

#### *AFFECTIONS OF THE PHARYNX.*

CHAP. I.—Angina Catarrhalis, . . . . . . . . . . . . . . 445
II.—Pharyngeal Croup, . . . . . . . . . . . . . . . 451
III.—Diphtheritic Inflammation of the Pharynx, . . . . . . . . 452
IV.—Phlegmonous Inflammation of the Pharynx, . . . . . . . 453
V.—Syphilitic Affections of the Pharynx, . . . . . . . . . 456
VI.—Retropharyngeal Abscess, . . . . . . . . . . . . 458
VII.—Angina Ludovici, . . . . . . . . . . . . . . . 459

### SECTION III.

#### *AFFECTIONS OF THE ŒSOPHAGUS.*

CHAP. I.—Inflammation of the Œsophagus, . . . . . . . . . . 461
II.—Strictures of the Œsophagus, . . . . . . . . . . . 463
III.—Dilatation of the Œsophagus, . . . . . . . . . . . 466
IV.—Morbid Growths of the Œsophagus, . . . . . . . . . 467
V.—Perforation and Rupture of the Œsophagus, . . . . . . . 469
VI.—Nervous Affections of the Œsophagus, . . . . . . . . 470

## SECTION IV.

### *DISEASES OF THE STOMACH.*

PAGE
CHAP. I.—Acute Gastric Catarrh, . . . . . . . . . . . . . 472
II.—Chronic Gastric Catarrh, . . . . . . . . . . . . 490
III.—Croupous Inflammation, . . . . . . . . . . . . . 501
IV.—Gastritis Phlegmonosa, . . . . . . . . . . . . . 501
V.—Inflammations from Caustics and Poisons, . . . . . . . 502
VI.—Chronic Ulcer of the Stomach, . . . . . . . . . . 504
VII.—Carcinoma of the Stomach, . . . . . . . . . . . 514
VIII.—Hæmorrhage from the Stomach, . . . . . . . . . . 521
IX.—Spasm of the stomach, . . . . . . . . . . . . 527
X.—Dyspepsia, . . . . . . . . . . . . . . . 531

## SECTION V.

### *AFFECTIONS OF THE INTESTINAL CANAL.*

CHAP. I.—Catarrhal Inflammation, . . . . . . . . . . . . 540
II.—Perforating Duodenal Ulcer, . . . . . . . . . . . 557
III.—Contractions and Closures, . . . . . . . . . . . 560
IV.—Scrofulous and Tuberculous Diseases of the Intestines and Mesenteric Glands, . . . . . . . . . . . . . . 572
V.—Carcinoma of the Intestines, . . . . . . . . . . . 578
VI.—Perityphlitis and Periproctitis, . . . . . . . . . . 581
VII.—Hæmorrhages and Vascular Dilatations, . . . . . . . . 583
VIII.—Colic, Enteralgia, . . . . . . . . . . . . . 591
IX.—Worms in the Intestines, . . . . . . . . . . . . 596
X.—Gastric Fever, . . . . . . . . . . . . . 604

## SECTION VI.

### *DISEASES OF THE PERITONÆUM.*

CHAP. I.—Peritonitis, . . . . . . . . . . . . . . . 609
II.—Ascites. . . . . . . . . . . . . . . . . . 620
III.—Tuberculosis and Cancer, . . . . . . . . . . . . 625

---

## DISEASES OF THE LIVER AND BILE-DUCTS.

## SECTION I.

### *DISEASES OF THE LIVER.*

CHAP. I.—Hyperæmia of the Liver, . . . . . . . . . . . . 627
II.—Suppurative Hepatitis, . . . . . . . . . . . . . 635
III.—Cirrhosis of the Liver, . . . . . . . . . . . . 640
IV.—Syphilitic Hepatitis, . . . . . . . . . . . . . 651
V.—Inflammation of the Portal Vein, . . . . . . . . . 653
VI.—Fatty Liver, . . . . . . . . . . . . . . . 655
VII.—Lardaceous Liver, . . . . . . . . . . . . . . 660
VIII.—Cancer of the Liver, . . . . . . . . . . . . . 662
IX.—Tuberculosis of the Liver, . . . . . . . . . . . 666
X.—Echinococci of the Liver, . . . . . . . . . . . 667
XI.—Multilocular Hydatids, . . . . . . . . . . . . 671

PAGE
CHAP. XII.—Hepatogenous Icterus, . . . . . . . . . . . . 675
XIII.—Hematogenous Icterus, . . . . . . . . . . . . 684
XIV.—Acute Yellow Atrophy of the Liver, . . . . . . . . 687

## SECTION II.

### DISEASES OF THE GALL-DUCTS.

CHAP. I.—Catarrh of the Gall-ducts, . . . . . . . . . . . . 692
II.—Croupous and Diphtheritic Inflammation, . . . . . . . . 695
III.—Obstruction and Closure of the Excretory Gall-ducts, etc., . . . . 696
IV.—Gall-stones and their Consequences, . . . . . . . . . 698

## DISEASES OF THE SPLEEN.

CHAP. I.—Hyperæmia of the Spleen, . . . . . . . . . . . . . 705
II.—Hypertrophy of the Spleen, . . . . . . . . . . . . 711
III.—Lardaceous Spleen, . . . . . . . . . . . . . . 716
IV.—Hæmorrhagic Infarction of the Spleen, . . . . . . . . . . 717
V.—Tuberculosis, Carcinoma, Hydatids, . . . . . . . . . . 720

## APPENDIX TO THE DISEASES OF THE SPLEEN.

CHAP. I.—Leuchæmia, Leucocythæmia, . . . . . . . . . . . 721
II.—Melanæmia, . . . . . . . . . . . . . . . 726

# DISEASES OF THE RESPIRATORY ORGANS.

## SECTION I.

## *AFFECTIONS OF THE LARYNX.*

### CHAPTER I.

### HYPERÆMIA AND CATARRH OF THE MUCOUS MEMBRANE OF THE LARYNX.

ETIOLOGY.—Catarrh consists in engorgement of the blood-vessels of any mucous membrane, accompanied by abnormal secretion, swelling, succulence of its tissues, and copious generation of young cells. Catarrh may arise from a purely mechanical hyperæmia of the mucous membrane; gastric and intestinal catarrhs are not unfrequently the result of compression of the portal vein; bronchial catarrh, the consequence of embarrassed flow into a diseased heart from the bronchial and pulmonary veins. We are therefore fully warranted in treating of hyperæmia and catarrh of the larynx and bronchi in the same chapter, provided only that we do not, like the laity, limit the term to that class of inflammations of the mucous membranes acquired by "taking cold," and to relieve which one wears flannel and drinks elder-blossom tea.

Now we find that liability to catarrh varies greatly among persons exposed to the same exciting cause; and that in one this mucous surface, in another that, is always the favorite point of attack. Special predisposition, in some cases, seems to coexist with a thin epidermis and a strong tendency to perspire. Badly-nourished, cachectic persons too, who are less capable of resisting the action of hurtful agents, are, on the whole, more prone to catarrh than full-blooded and robust individuals. In other instances there is no clew whatever to the cause of an intense predisposition to this affection. Effeminate habits seem to aggravate it. At all events, we see that country people, shepherds, and others, who live continually exposed to changes of temperature and to stress of weather, are less frequently thus affected than persons of sedentary habits, and those who are but rarely subjected to such exposure.

The fact, also, is inexplicable, that an agent which, in one subject, will almost always cause catarrh of the larynx, in another will, as invariably, give rise to coryza, catarrhal diarrhœa, or to a bronchial catarrh. Of one thing alone we may be sure—that, after repeated attacks, the laryngeal mucous membrane remains more vulnerable, becomes a weak place, as it were, and that trifling causes serve to excite fresh disease of the organ.

The exciting causes are—first, local irritants which act upon the larynx. Among these are the breathing of very cold air; the inhalation of dust and acrid vapors; loud screaming too, and shouting, singing, and violent coughing. In the latter procedure the air is driven forcibly through the narrow chink of the glottis, causing severe friction upon its free edges, an injury quite as great as that occasioned by other agencies. If acrid liquids or hot water penetrate into the organ, the most intense form of laryngeal catarrh ensues.

Secondly, chilling of the skin, particularly that of the neck and feet, will give rise to this disease. We are constantly seeing some one who, having left off his neckcloth or woollen stockings, suffers next day from laryngeal catarrh. Difficult as it may be to give a physiological explanation of the occurrence, a genetic connection between the two events is not to be denied.

Thirdly, catarrh not unfrequently spreads from neighboring organs to the laryngeal mucous membranes. We often see it extend thus from the nose or bronchi without the supervention of any new irritant. The pharynx is sometimes the point of origin. This is especially the case in that form of the malady induced by the abuse of spirituous liquors, which have a direct action upon the pharyngeal mucous membrane. Indeed, we may often notice a hoarse, stridulous voice, or other sign of acute laryngeal catarrh, coming on in consequence of a debauch, although the person affected may not have shouted or sung much. Habitual topers almost always have catarrh of the pharynx, in which the laryngeal mucous membrane takes part.

Fourthly, laryngeal catarrh is a common symptom of constitutional disease resulting from infection or contagion. Among the acute affections, measles and exanthematic typhus; among the chronic, syphilitic disorders are the maladies especially prone thus to localize themselves upon the larynx. We still lack a thorough insight into the physiological connection between the blood-changes at the root of the disorders and the nutritive derangements visible upon the skin and mucous membranes in these complaints.

Fifthly, those cases in which the disease forms a portion of a very extensive and severe catarrhal affection, the "grippe," or influenza. In its onset, in its extent, and in the severe constitutional disturbance which accompanies it, this epidemic bears great resemblance to the acute exanthemata. In influenza, catarrh must be regarded as a constitutional if not an infectious disorder.

Sixthly and finally, morbid growths and ulcers, particularly tuberculous ulcers of the larynx, are accompanied by a catarrh. These symptomatic cases, which, like the hyperæmia about ulcers and carcinomata of the skin, are subject to exacerbations and remissions, form important exponents of certain conditions, and in particular account for the fluctuations in the symptoms of ulceration and malignant growths of the larynx.

Anatomical Appearances.—In acute catarrh of the larynx, the mucous membrane of the cadaver does not always reveal a degree of redness and vascular engorgement such as the violence of the symptoms during life would lead us to expect, and such as could then be demonstrated by laryngoscopic observation. This is due to the richness of the laryngeal mucous membrane in elastic fibres, which, remaining extended by the blood contained in the vessels during life, after death contract, and expel the contents of the capillaries. However, in very violent catarrhs, apoplexies occur (ecchymoses) in the substance of the mucous membranes, which after death present either a mottled or a uniformly reddened aspect.

On the surface of the mucous membrane the cylindrical-form ciliated epithelial cells, which constitute the most superficial layer of the stratified epithelium of the larynx, are wanting in places; but under the microscope we find in the slightly-turbid serum which adheres to the mucous membrane numerous transparent cells, for the most part unilocular, and which are detached young epithelial cells of the deeper layers, or of the mucous follicles, and are called mucous corpuscles. The substance of the mucous membrane itself is swollen, moister, and flabby. The submucous tissue may exceptionally be the seat of considerable serous infiltration, a condition to be treated of by-and-by as œdema glottidis.

In chronic laryngeal catarrh the mucous membrane appears more or less dark, dirty bluish-red, or brownish (from deposit of pigment in consequence of previous ecchymosis). The vessels are sometimes varicose and gorged with blood, the flabby mucous membrane having lost its elasticity. Its tissue is generally thickened, firmer, and hypertrophied. The surface appears, in many places, uneven and granulated, from the tumefaction and distension of innumerable mucous follicles which exist in the larynx. Sometimes it is covered by a scanty glairy mucus; sometimes with profuse yellow secretion. The young epithelial cells, upon the copious admixture of which the opacity and yellow color of this "mucopurulent" secretion depend, are indistinctly granulated, and their nuclei are often divided. They are quite analogous to the young cells found in abscesses. We now no longer call them mucous, but pus corpuscles, although it is not easy, in all cases, to distinguish between the two forms.

The anatomical changes which the laryngeal mucous membrane undergoes can be better ascertained by means of the laryngoscope during life than by *post-mortem* dissection. We find that catarrh of the larynx does not always involve the whole surface of the organ, but is often confined to particular sections. Thus there may be catarrhs, limited to the region of the epiglottis, the arytæno-epiglottidian fold, the arytenoid cartilage, the true or the false vocal chords. There is one very interesting form of circumscribed swelling of the membrane between the arytenoid cartilages and a little beyond, which *Lewin* has repeatedly ascertained to be a cause of the chronic hoarseness among officers who have been much in the habit of shouting the word of command.

Apart from the catarrhal sores, and the polypous growths of which we are to treat in a separate chapter, chronic laryngeal catarrh, and chiefly that form of it which accompanies syphilitic and tuberculous ulceration, sometimes leads to a thickening and induration of the sub-mucous tissues. Its metamorphosis into a brawny fibrous mass, which often has the effect of greatly contracting the larynx, and rendering the vocal chords stiff and immovable, takes place in a manner quite analogous to that in which induration of other organs arises, especially of the stomach.

SYMPTOMS AND COURSE.—Acute catarrh of the larynx rarely begins with shivering; indeed, in most cases, when it does not spread into the bronchi, catarrhal fever is also absent throughout the whole course of the attack. The general condition is untroubled, and the symptoms of disease alone refer to the functional derangements of the suffering organ.

The patients first complain of a feeling of titillation, or, in more severe cases, of a sensation of burning or soreness in the throat, which is aggravated by speaking or coughing. Sensation in these parts extends as far down as the bronchi of the second magnitude. If, therefore, the covering of the epiglottis, the arytenoid, or arytæno-epiglottic ligament be the seat of catarrh of some intensity, deglutition becomes painful. In addition to these symptoms, alteration of the voice furnishes a characteristic sign of disease of the larynx. The voice grows deeper, hoarse, cracked, and may finally become inaudible. The vocal sounds, as we all know, originate entirely in the larynx, which is formed like a reed-pipe, with membranous reeds. In the act of speech, the lower vocal chords approach one another so closely as to project into the organ as vibrating membranes. If thrown into vibration by the current of a strong expiration, a tone results whose elevation or depth of pitch depends upon the degree of tension in which they are set. Now, we have stated above that in catarrh of the larynx the mucous membrane swells up and becomes relaxed, and is covered by a secretion in more or less profusion. If the vocal chords also become involved in this condition, the tension to which the laryngeal muscles can bring them is inadequate so to increase the frequence of their vibrations as to produce a tone of a pitch such as would result at the same tension in normal chords. Thus the voice is rendered deeper. From the irregular swelling, and from the presence of the mucus which covers the chords and makes their surface uneven, the voice is hoarse and its tones are false (just as the note of a violin-string is altered when smeared with grease). At last the swelling and relaxation of the chords may so increase that the utmost tension to which the muscles can bring them is insufficient to allow of sonorous vibration. The voice then is noiseless or extinct. Extinction of the voice may also depend upon swelling of the false vocal chords, as the latter, when thus brought into contact with the true chords, render their free vibration impossible. Frequently the hoarse voice of the patient suddenly breaks into discord, or "cracks." This occurs when the tumid chords, bathed in mucus, momentarily touch one another, so as to produce vibratile nodes, a great increase in the frequence of vibration, and consequent elevation of pitch in the tone.

Besides the titillation, burning, and hoarseness, there is also violent cough. In the healthy larynx we see coughing-spells provoked as reflex phenomena upon exposure of the organ to any undue irritation—the intrusion of a foreign body, for instance. When the laryngeal mucous membrane has become the seat of catarrh, we find that analogous fits of coughing are excited by the most trifling and unobservable causes, and, to all appearance, spontaneously. Sometimes, in these paroxysms, so severe a spasm besets the muscles of the glottis that, in the inspiratory movement by which the fit commences, the air can pass but slowly through the contracted rima glottidis, and with a wheezing sound, while the expiratory effort consequent upon this long-drawn, sonorous inspiration, is only capable of effecting momentary openings of the constricted passage, thus producing the interrupted, rattling, short "hacks" of a cough. The effect of vigorous expiration through a narrow glottis, such as we make in blowing upon wind instruments or in straining, is to compress the thorax, and thus to check the influx to it of blood from the veins, so that the jugulars distend and the face grows red, or even bluish. In other cases of long standing the tone of the cough likewise deepens, and grows harsh and hoarse from thickening and unevenness of the chords. Quite frequently, from some powerful expiratory effort, the thickened chords are made to bulge upward and are thrown into strong tension. The hoarse cough then changes into a "bark," or, upon momentary contact of the chords, the sound of the cough is cracked.

When catarrh is confined to the larynx, the expectoration is scanty.

At first it is either absolutely wanting, or else it is clear or glairy. This mucous sputum, "sputum crudum" of the ancients, does not often contain detached ciliary epithelium. There is much more commonly a moderate admixture of young cells from the deeper layers of the epithelial covering, or of mucous corpuscles from the follicles. As the disease progresses and begins to abate, the expectoration becomes thicker and more rich in young cells, which rather resemble pus-cells. This form of expectoration, the "muco-purulent," is the "sputum coctum" of the older physicians.

As the submucous tissue is seldom much swelled or infiltrated, save in rare cases, dyspnœa in simple acute laryngeal catarrh of adults is of exceptional occurrence. In grown persons the glottis, especially the posterior part of it, the pars respiratoria of *Longet*, is a tolerably roomy triangular space, bounded by the base of the arytenoid cartilage, and does not become impervious to air by reason of swelling of its mucous membrane alone. Even among children it is rare for the dyspnœa of simple laryngeal catarrh to be continuous. Although in the latter the glottis is smaller, its entire space forming but a narrow chasm, yet the swollen chords are generally separated from one another by the free action of the posterior crico-arytenoid muscles, which act with every inspiration, so that there is no hinderance to the entrance of air. A glance at the laryngoscope is enough to convince any one that the rima glottidis gapes during inspiration so widely that a moderate swelling of the mucous membrane cannot materially obstruct the passage of air or produce symptoms of dyspnœa. In certain well-authenticated cases of intense catarrhal laryngitis, however, there has been such serious swelling of the true chords, or of the false ones, which cannot be drawn asunder by muscular action, as to place the patient in danger of suffocation.

Not at all rarely we see a child who has been coughing a little during the day, and been hoarse without feeling ill, wake up suddenly in the night with great oppression of breathing. The inspiration is troublesome and protracted; the terrified child throws himself about in bed, or springs up, clutches anxiously at the throat; the cough is hoarse and barking. These attacks, which are often confounded with croup, or called pseudo-croup, usually vanish completely after a few hours—often much sooner. It is to these that the warm milk, the hot sponge laid upon the throat, the judiciously-administered emetic owe their reputation as panaceas against croup, sure to cut it short if given in time. One might suppose that these accidents arose from an unusually aggravated but transient swelling of the mucous membrane, and from a narrowing of the glottis, which could not be compensated for by muscular action, just as in coryza we see sudden absolute closure of one or other of the nostrils; or we might think that a spasmodic closure of the glottis had allied itself to the mucous irritation as a reflex symptom, such as we shall describe by-and-by as laryngismus stridulus. But there is another and more probable explanation. These attacks take place almost solely during sleep, pass off after the child has cried, coughed, and vomited awhile, to recur almost as soon as he falls asleep again. Hence it is likely that the dyspnœa is occasioned by a collection of tenacious secretion in the glottis, and perhaps also by a dryness of the rima glottidis, which tends, as it were, to glue it up. At all events, this explanation likewise accounts for the action of the remedies alluded to above, and so justly prized. Attacks like these are often repeated for several successive nights, while during the day the children play about gayly, and, with exception of a slight cough, seem perfectly well.

As regards the course, duration, and termination of laryngeal catarrh, the sputa cocta generally appear after a few days, when the sensitiveness of the larynx, the hoarseness, and the cough abate, the disease terminating by recovery at the end of about a week. In other cases the complaint lasts for several weeks. During the day the patient is somewhat hoarse, but otherwise is well, save that in the mornings and evenings he is troubled by violent and protracted fits of coughing. The sputa remain crude, until at last, often upon some change of weather, the disease subsides. In other cases again, after repeated relapses, chronic laryngitis is the result. A fatal termination, uncomplicated by any other cause of death, is one of the greatest of rarities.

*Chronic Catarrh of the Larynx.*—As, even in acute catarrh, the sensitiveness of the mucous membrane abates with the appearance of the sputa cocta, so in the chronic form we hardly ever find titillation, burning, or soreness in the larynx. Hypertrophy of the mucous membrane, however, and continued thickening of the vocal chords, as described in the last section, produce a permanent deepening of tone in the voice, and render it harsh and hoarse.

In the cases alluded to above, in which the mucous membrane of the posterior wall of the larynx close below the vocal chords is thickened and swollen, hoarseness proceeds from the intrusion of a fold of membrane between the posterior adjacent surfaces of the chords, whenever the voice is raised, so that the glottis cannot contract properly. This chronic hoarseness, the result of repeated acute attacks, is the chief and often the sole symptom of chronic laryngitis. A cracked voice usually accompanies long-standing chronic hoarseness. From time to time, a slight aggravation or some acute irritation of the mucous membrane so thickens the vocal chords, as to render the voice quite inaudible.

In many instances, besides the aphonia, there is also a periodical spasmodic cough, such as we have described as symptomatic of acute laryngeal catarrh, although the attack seems to proceed rather from an accumulation of secretion in the pouches of Morgani, and passes off after the expectoration of a small quantity of peculiar lumpy yellowish mucus. Here too, for reasons already given, the cough may be harsh, hoarse, and grating, swelling into a bark.

The addition of a whistling, stridulous inspiration and expiration, as it certainly does not proceed from mere swelling and hypertrophy of the mucous membrane, denotes the presence of a complication—either thickening and induration of the submucous tissue above described, or a morbid growth encroaching upon the cavity of the larynx, or else syphilitic laryngitis.

It is only by means of the laryngoscope that we can determine positively with which of these three main forms of laryngo-stenosis we have to deal. On the other hand, the association of fever, emaciation, and night-sweats, with chronic laryngeal catarrh (catarrhal laryngeal phthisis being rare), should awaken our suspicion of latent disease of the lungs, and induce repeated physical exploration of the chest.

The course of chronic laryngitis is usually tedious. It is only by most cautious and judicious treatment that a cure can be effected, and there is nearly always a strong tendency to relapse.

DIAGNOSIS.—Catarrh of the nose and that of the pharynx, which also produce alteration in the voice, are easily distinguishable from catarrh of the larynx. In the former, the resonance of the contracted nasal and oral cavities, the "timbre" of the voice, is changed—the speech is nasal or guttural; in the latter, the tone itself is modified, the voice is deeper, cracked, and hoarse.

Confusion with croup is more apt to occur. To anxious mothers, mere hoarseness and a barking cough furnish evidence enough of the presence of that dread disease, even although the child may be well enough otherwise. The addition of a nocturnal paroxysm of dyspnœa will often mislead the physician himself. Thus it is that we so often hear of children who have suffered eight, ten, or even more, attacks of croupous laryngitis. Croup is neither so frequent nor so innocent a disease as to admit of the likelihood of such frequent recoveries. In most of such cases there has been error of diagnosis. The points of distinction between the two diseases will be more fully given while treating of croupous laryngitis. For the present we call attention to one point only, upon which the laity lay greater stress than the profession. Nasal catarrh is almost as surely symptomatic of the catarrhal form of laryngitis as is croupous pharyngitis of true laryngeal croup. The satisfaction of mothers at the "running nose" of their child is well founded—the rarity with which simple catarrh is complicated with graver disease having given rise to the old custom of salutation after sneezing.

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The distinctive points between chronic simple catarrh and ulceration or growths in the larynx are to be discussed hereafter.

**Prognosis.**—The prognosis both of acute and chronic laryngitis becomes apparent from the foregoing sketch of its symptoms. When uncomplicated, the disease is seldom fatal. The prognosis as to complete recovery is favorable in the acute disease, although a tendency to relapse remains; in chronic cases, it is more unfavorable. Induration of the submucous tissue is incapable of resolution.

**Treatment**—*Prophylaxis.*—It is advisable, rather cautiously to habituate children to the causes of this disease than to enervate them by a systematic over-protection which tends to increase the liability to its attacks upon every trifling occasion. Do not shut up little children in the house, even though they have suffered from laryngitis; but keep them in the open air.

In bad weather, let them be warmly clad; but their necks should never be overheated with thick woollen shawls, etc. A silk ribbon worn about the neck has the reputation of a sympathetic prophylactic. Washing of the throat in cold water, and cold river and sea bathing, cannot be sufficiently commended. They form the best of prophylactics; but, in prescribing their use, the most definite and rigid rules must be laid down as to time, duration, and temperature. The more precisely we direct, so much more punctually do patients obey.

*Indication as to Cause.*—If the cause of the catarrh be the direct action of some irritant upon the mucous membrane, the patient must be protected from its further influence. To guard the affected larynx from further irritation, let the patient be kept in a uniform temperature, regulated by the thermometer. Forbid all loud and continued talking or singing, and, above all, urge the patient to resist the inclination to cough. Even though not entirely successful in this, yet much may be gained by determination on his part. The assertion that he cannot help coughing should never deter you from persistently telling him *not to cough.* In violent paroxysms, which, although consequent upon the catarrh, are active causes of its perpetuation, the common "cough-drops" and syrups containing antimony are useless. If the remedies mentioned above prove inadequate, we must resort to the narcotics. True, great caution in exhibiting these agents is demanded in treating children, but among adults they certainly have not been used with proper boldness and freedom. It is surely more reasonable, and indubitably more efficacious, to prescribe ten grains of Dover's powder at night, or occasional small doses of morphia (morphiæ, gr. j; aqua laurocerasi, ʒij; gtt. x every three hours) to a patient with severe laryngeal cough, than to plague him with liquorice, caramel, sulphuret of antimony, and the like.

When chilling of the skin, feet, or throat is the cause of catarrh, diaphoresis is indicated. The best and simplest of diaphoretics are hot elderbloom-tea, and warmth in bed, warm foot-baths, wrapping the throat in a woollen stocking, sinapisms repeated from time to time, or hot poultices, the chilling of which must be carefully prevented. Envelopment of the entire body in cloths wrung out in cold water, the use of brief cold foot-baths, the application of a cold, stimulating compress to the throat, have a similar action to that of the above-mentioned procedures, and are to be regarded as local excitants, or cutaneous stimulants. The merits of such measures are overrated by the hydropathists. They are, nevertheless, quite useful in the hands of persons skilled in their employment, and who are generally enthusiastic in their favor.

In cases where the catarrh has extended from the fauces into the larynx, the causal indications are for astringent gargles, and for pencilling the throat with a solution of nitrate of silver or alum.

The indication, from the disease itself, in acute laryngeal catarrh, uncomplicated by œdema glottidis, never requires either local or general bloodletting, for, although the books give detailed direction for their use in most cases, it is quite sufficient to produce a determination to the skin, by means of cutaneous stimulants, and thus to reduce the mucous hyperæmia. In fact, the case usually does well without any treatment whatever, or even when mismanaged. Tincture of pimpinella, which has the reputation of a specific, may be tried, but is not much to be relied upon. For a drink, we may give seltzer-water, either pure or mixed with equal parts of hot milk.

Quite empirically, it has been found that greasy materials are hurtful, while strongly salted ones act beneficially upon acute laryngeal catarrh. An unsoaked herring is a well-known popular remedy, and may, perhaps, set up a derivative action upon the mucous membrane quite as powerful as that produced by a sinapism upon the skin of the throat.

For chronic laryngeal catarrh, Plummer's pill (calomel, with sulphuret of antimony), combined with belladonna, or hyoscyamus, used to have the name of a specific. The latter ingredients might be of use in moderating the coughing fits. Whether they are more effectual than opiates may be doubted. The sulphuret of antimony is superfluous; the calomel, in catarrhal inflammation, objectionable. Instead of the more moderate irritants, as sinapisms, and the like, in chronic laryngitis, we employ stronger derivatives. The most common is croton-oil, either alone or combined with five parts of oil of turpentine, which is to be rubbed over the skin of the larynx for several days in succession, until vesicles and pustules appear.

The use of the alkaline muriatic mineral waters (Säuerlinge, Halloid salts) has an unmistakable influence upon the course of many cases of chronic laryngeal catarrh, which, unfortunately, cannot as yet be distinguished from cases in which it fails. For this mode of cure, it is best to send the patient to such places as Ems, Obersaltzbrunnen, or Gleichenberg, and only when his means will not permit him to do otherwise, to allow him to use seltzer-water, or one of the so-called mineral waters as a cure, at home. We may let him drink the Ems or Kesselbrunnen water, or the Krähnchen of Ems, on the spot, as they have, respectively, a temperature of 117° F. and 90° F., without the addition of warm milk or warm whey. In order to warm them, it is better to mix the Obersaltzbrunnen, or the imported Ems-water, with equal parts of hot milk. That the far more customary addition of whey should have any real advantage over that of milk, is, at least, doubtful. The "well-prepared whey," at celebrated watering-places, furnished generally by a "Swiss," and, if possible, by an Appenzeller, in his national costume, so much lauded in the newspapers and bath-journals, and to which often more credit is given than to the springs themselves, is merely milk, minus cheese, and can hardly effect more than the milk from which the cheese has not been eliminated. It is only in the somewhat rare cases, in which milk is not well borne by the patient, while the whey is borne well or better, that I allow the latter to be added to the mineral water instead.

Several hypotheses have been advanced as to the action of the alkaline-muriatic mineral waters. The fact that the ashes of the mucus are richer in salt (chloride of sodium) than the ashes of the blood, and that mucus becomes less tenacious upon the addition of salt, seems certainly to indicate that salt plays an important rôle in the formation of mucus, but it by no means justifies the conclusion that the use of salt effects a cure, or more rapid resolution of a catarrhal process.

In other quarters (*Sprengler*) the principal importance has been attributed to the amount of alkaline carbonates contained in these mineral waters, and, depending upon an observation of *Virchow's*, according to which very dilute solutions of alkalies are capable of exciting the ciliary movements in epithelium, they assert, in explanation of the beneficial action of the waters in question, that their use reëstablishes the extinguished or repressed ciliary vibrations. Grave objections may be brought against this explanation of the action of the saline waters, which is not merely palliative, but in many cases absolutely curative, and we must be content with the empirical fact, that the springs of Ems, Obersaltzbrunnen, and Selters, have often alleviated or cured chronic laryngeal catarrh. The cold sulphur springs, too (such as those of Weilbach, in the dukedom of Nassau, of Eilsen, in the principality of Schaumburg-Lippe, of Langenbrücken, in the grand-dukedom of Baden), which we usually make use of, like those of Obersaltzbrunnen, and Seltzer, mixed with warm milk, or whey; the sulphur springs, also, of the Pyrenees, above all the Eaux-bonnes, are, with good reason, in repute, in the treatment of chronic laryngitis. Our conjectures as to the *modus operandi* of these waters are as yet vague and untenable—a matter, however, far less to be regretted than the fact that we have no criterion whereby to predetermine the cases in which relief may be expected, and those in which they do no good.

In obstinate and inveterate cases of chronic laryngitis, local treatment deserves an extended trial.

The attempt to blow medicated powders into the larynx is an ancient practice. For this purpose, a long quill, or a glass tube, eight or ten inches in length, and several lines in diameter, is employed. A few grains of the powder to be inhaled is laid within one end, the other end is introduced as far as possible into the mouth of the patient, who is then to close his lips, and to draw a deep inspiration, or else we may blow into the external end of the tube. If this procedure should excite violent inclination to cough, we may assume that a part of the medicament, at least, has reached its destination, although, no doubt, the greater part remains clinging to the velum-palati and pharynx. The medicines most frequently used in this practice by *Trousseau* are arg. nitrat. (gr. j—ij to sacc. alb. ʒ j—ij), calomel (gr. x—xx to sacc. ʒ j—ij), alumen. (ʒ ss—j to sacc. alb. ʒ ij). At present, by aid of the laryngoscope, and of a curved tube, inserted as far as the entrance of the larynx, we can blow into it almost the whole of the powder.

Another procedure, which acts with tolerable certainty, consists in expressing the contents of a small sponge, made fast to the end of a little rod of whalebone, and saturated with solution of arg. nit. (gr. xx to ℥ j) over the entrance of the larynx. The result of this mode of treatment is often both instantaneous and brilliant, and finds a striking analogue in the efficient use of solution of nitrate of silver in the treatment of catarrhal conjunctivitis.

An adept in the use of the laryngoscope enjoys the great advantage of being able to assure himself by direct ocular observation of his success in passing the sponge behind the epiglottis.

The most recent and generally-employed method of producing the direct action of medicaments upon the mucous membrane of the larynx consists in causing the patient to inhale them in solution reduced to the condition of a spray or mist. The apparatus hitherto employed for this purpose consequently bear the names of néphogène, pulverization, [nebulizer], inhalations apparatus. Of these there are two kinds. In one, the slender stream of liquid to be inhaled is driven forcibly against a small convex disk, and thus reduced to the condition of spray, as in the machine of *Salès-Giron* and its modifications by *Waldenburg*, *Lewin*, and *Schintzler*. In the second sort, small quantities of the liquid for inhalation are "nebulized" by the action of a jet of compressed air. The néphogène of *Matthieu* is thus formed, as well as the more simple and cheaper hydroconion of *Bergson*, which I formerly used at the clinique. By the happy ingenuity of *Siègle* the apparatus of *Bergson* has been so modified as to substitute steam for the current of compressed air, which nebulizes the medicament. This cheap instrument of *Siègle*, with its various modifications, consisting chiefly in the exchange of the fragile retort of the original for a small boiler of tinned brass, for the production of steam, has such advantages over all other inhalation machines, as to have almost universally superseded them. At my clinique, instruments made on *Siègle's* principle are the only ones in use. The controversy, as to whether the liquid inhaled actually penetrates into the air passages, has been decided. The fact is beyond all doubt. In recent catarrhs, with scanty and tough secretion, it is best to use a solution of sal-ammoniac or of common salt for inhalation (gr. x—xx. to ℥ j). In catarrh of longer standing, in which the secretion is more copious and muco-purulent, a solution of alum (gr. v—x : ℥ j), tannin (gr. ij—x : ℥ j), argent. nitrat. (gr. i—x : ℥ j). During inhalation of the latter, in order to avoid staining the patient's face, a mask must be used, or else an appliance such as accompanies the apparatus which we employ. I am unable to state, from my own experience, whether the inhalations of narcotic solutions (morphiæ acet. gr. $\frac{1}{8}$—$\frac{1}{4}$ : ℥ j), (tr. opii gr. ij—iv : ℥ j), (ext. hyoscyami gr. ss—j : ℥ j) are of any material service in allaying the impulse to cough.

By exaggerated praise of the treatment by inhalation, a discovery of real value has been retarded rather than advanced, and indeed has been often brought into positive discredit in the estimation of discreet and thoughtful men, who have failed in verifying the brilliant success claimed for it in treatment of the various diseases of the air-passages. However, "in emptying the bath, one need not spill the baby." The introduction of the inhalation apparatus does not mark a new era in therapeutics; nevertheless, obstinate, inveterate pharyngeal and laryngeal catarrhs, which hitherto have resisted all modes of treatment, are now frequently cured, after persevering inhalation of a solution of alum or of nitrate of silver.

Spray-baths and inhalation-rooms have been established of late in many well-known watering-places, particularly at the "brine-baths" (*Soolbädern*). The most simple baths of brine-spray are the promenades and galleries along the salt-works of Kreutznach, Koesen, Elmen, and Reichenhall. The atmosphere there is heavily charged with a weak solution of chloride of sodium. At Kreutznach and Reichenhall the brine is also nebulized in appropriate closets, after the method of *Salès-Giron.* In Rehme (Oeynhausen), a salt-spring, there is an excellent spray-bath. At Kreutznach, Reichenhall, and Ischl, besides the spray, the warm steam generated by the boiling brine is also inhaled. This contains less salt than the spray of the salt-works, and inhalation hall. Whether, and how, inhalation of the brine-spray acts as a remedy for laryngeal catarrh, is still a question. Many patients, especially those in the closets, complain of pain in the eyes, and contract a conjunctivitis by the same process, whereby they hope to be relieved of laryngeal or bronchial disease, a fact which has many analogues, should further experience pronounce in favor of inhalation of brine in chronic laryngitis. In proposing the inhalation of brine in this disease, the fact that it contains iodine and bromine has also been borne in mind. The momentary relief, obtained by the patient during, and for a short time after, inhaling, is attributable simply to the liquefaction of the mucus in air-passages by the nebulized liquid, whereby, in the narrowest sense of the word, the cough is rendered "looser." At Ems the thermal gases, and at the sulphur springs, the vapor of the richly sulphureted waters, have recently been similarly inhaled. With regard to the latter, it may be remembered that even Galen recommended an abode near Vesuvius to the "phthisical," that they might respire the moist sulphureted vapor as it rose.

The diet for chronic catarrh of the larynx must be similar to that for the acute; salted articles, indeed, particularly the roe of a herring taken fasting, are in especially good repute.

Besides allaying the cough, the symptomatic indication calls for a means of promptly relieving the nocturnal paroxysms of dyspnœa above described. To apply leeches to the throat, though often done, is useless. The repeated application of a sponge dipped in hot water to the throat, until the skin grows red, the exhibition of copious draughts of hot liquid, and, above all, the administration of an emetic, are often indicated and frequently are of surprising efficacy. As an emetic, ipecacuanha, or tartar emetic, is to be preferred in these cases to sulphate of copper, and should be given in efficient doses (best according to Hufeland, pulv. rad. ipec. ℈j, ant. et pot. tart. gr. j, scillæ. oxymel ʒiij, aqua ℥j ss; shake well, a teaspoonful every ten minutes).

Should the paroxysm recur, the emetic is to be repeated. It is a good rule not to let the child sleep too profoundly, but, from time to time, to waken it and let it drink. This will often cause it to expectorate, and we thus prevent the accumulation and drying up of the secretions in the rima glottidis.

## CHAPTER II.

### CROUP.—ANGINA MEMBRANACEA.—LARYNGITIS CRUPOSA.—MEMBRANOUS CROUP.

ETIOLOGY.—Croupous inflammations are inflammatory disorders in which a fibrinous exudation which rapidly coagulates is thrown out upon the free surface of a mucous membrane, but which involves the epithelium only. If the croup-membrane thus formed be detached, the epithelium is quickly reproduced. No loss of substance occurs in the mucous membrane itself, and no scar remains. The diphtheritic process is also characterized by the production of a fibrinous rapidly-coagulable exudation, but differs from croup, the exudation forming, not merely upon the surface of the mucous membrane, but also within its substance. The pressure upon the blood-vessels exerted by this interstitial exudation, as well as by the swollen elements of the tissue, results in sloughing of a portion of the inflamed mucous membrane, and in the formation of a so-called diphtheritic eschar, which, upon separating, occasions a loss of substance and consequent cicatrix. Of these two forms of inflammation (the essential duality of which has of late been much in dispute), it is almost exclusively the croupous form which appears in the mucous membranes of the respiratory passages; and it is only in rare and solitary instances of secondary croup, when that malady forms part of some general acute infectious disorder, as measles, small-pox, typhus, scarlatina, or epidemic diphtheria, that a transition from croupous to diphtheritic inflammation is observable. Even here, too, though the pharynx may be the seat of a most exquisite diphtheria, it is far more common, and it is, in fact, the rule, for the laryngeal inflammation to retain the characteristics of true croup. (See chap. "Diphtheria.")

Croup is of far rarer occurrence upon other mucous membranes than upon those of the air-passages, and, during childhood, is almost exclusively a disease of the trachea and larynx, rarely affecting the alveoli of the lungs. On the other hand, croupous pneumonia, a true croup of the air-cells, is one of the most common diseases of adults, in whom primary croup of the trachea and larynx scarcely ever occurs.

Although peculiarly a disease of childhood, still the disposition to it is less during the period of suckling. After the second dentition, too, the disease is more rare; so that the period of greatest predisposition for croup lies between the second and the seventh year of life. Boys are more subject to it than girls; but it is an error to suppose that vigorous, full-blooded, blooming children are especially liable. On the contrary, tender, delicate, ill-nourished offspring of tuberculous parentage, with pale skin and conspicuous veins (an ominous sign even for the laity), children with a tendency to moist eruptions, to enlarged lymphatics, or to acute hydrocephalus, suffer from croup with equal or even greater frequence than those who are more robust. It is our daily experience that, in the great mortality which desolates certain families, a portion of the members die of croup, and another of hydrocephalus, while, in the survivors, pulmonary tuberculosis develops later in life (see "Pulm. Tuberculosis"). It would appear that the croup not unfrequently begins very soon after the disappearance of a moist eruption on the head or face.

The croup is more common in northerly, windy, damp places, bordering on the water, than in southerly, warmer, and more protected regions. Not unfrequently we observe its epidemic appearance. At such times many children are attacked even in one small place, and often several children of the same family in quick succession, and by the most intense and pernicious form of the disease.

It is this epidemic croup of the larynx which seems most commonly to be combined with croup of the pharynx. In some croup-epidemics facts have been observed which make it somewhat probable that the disease may spread by contagion. It is questionable, however, whether there may not have been confusion with that highly-contagious malady, epidemic diphtheria, in these cases, as we shall hereafter demonstrate the fact that secondary croup of the larynx often accompanies diphtheria of the fauces.

The exciting causes of croupous laryngitis are in most cases not to be explained. Sometimes the irritated condition of the mucous membranes, known as "a cold," occasions the disease. A sharp northerly or northeasterly wind stands in especially evil repute in this respect. We shall treat hereafter of the relation of secondary croup to the infectious diseases.

ANATOMICAL APPEARANCES.—The affected mucous membrane shows a varying degree of reddening, partially through ecchymosis, and in part through injection. It has been maintained that the redness diminishes when the exudation increases; nay, formerly the opinion prevailed, based upon the absence of inflammatory reddening in croup, that it constituted a peculiar form of inflammation wherein there was no hyperæmia. We have already shown that the pallor, after death, of the mucous membrane, which during life had been hyperæmic, is principally due to the abundance of elastic fibres in its tissues.

The mucous membrane is deprived of its epithelium, and, together with the submucous tissue, is swollen and relaxed. Even the muscles of the larynx seem moist, pale, and softer. Very often too, but not always, the mucous membrane of the cadaver is still covered with exudation. The frequent absence of the croup-membrane, in the bodies of persons who have died with all the symptoms of croup, has given rise to an artificial division into true and false croup, and even to-day there are many physicians who maintain that, in the subjects in which, upon autopsy, no membrane has been found in the larynx, the cause of death has not been true croup.

In croup, too, a fluid plasma first exudes, and it, of course, does not coagulate until after exudation. If ejected from the body, either in the coagulated or liquid form, on section we find the larynx to be free; but we have to do with exactly the same disease as that in which a coagulated coating is found upon the mucous membrane. Croupous exudation sometimes has the consistence of a thick cream; sometimes it forms a compact, tough membrane; sometimes it entirely lines the interior surface of the larynx as a continuous sheet, and is prolonged into the trachea and even into the bronchi, forming tubular and ramifying clots; sometimes it only presents isolated flakes and patches, which cling here and there to the mucous membrane.

The softer and thinner pseudo-membranes may generally be detached from the mucous surface with ease; the tougher and more coherent ones cling more firmly. Upon the external surface of this firm, strong substance, which is often more than a line in thickness, we frequently may notice numerous red streaks, and points of adherent blood, which correspond to small bleeding spots of the mucous membrane upon whose areolar layer the exudation is situated.

After persisting for a longer or shorter time, the pseudo-membranes gradually become loosened by a serous exudation which proceeds from the mucous surface, and are expelled either in the form of continuous tubes and sheets, or in small flakes and patches.

Under favorable circumstances, the epithelium is soon reproduced, and the laryngeal mucous membrane returns to its normal condition. In other cases, a fresh membrane succeeds upon the fall of the first one, and thus the process may be many times repeated, until the disease exhausts itself, or until the patient succumbs.

The membrane of croup consists microscopically of amorphous or finely-fibrillated fibrin, in which numerous young cells have been entangled during the process of its excretion.

The frequent association of pharyngeal croup with croup of the larynx has a very important bearing, not only upon the diagnosis of the disease, but also upon the physiological elucidation of its symptoms. The French do not acknowledge any case as true croup, where this complication is absent; calling all others false croup. Since attention has been called to the subject in Germany, it has been found that the coexistence of both forms of the malady, although extremely frequent, is by no means constant.

In the bodies of croupous children we may almost always find intense hyperæmia of the lungs and bronchial mucous membrane, bronchial catarrh with copious secretion, œdema of the lungs, and not uncommonly croup of the bronchi, spots of pneumonia, atelectasis, with both vesicular and interstitial emphysema. It will be shown, hereafter, that such conditions are in a great measure the necessary consequences of laryngeal croup.

SYMPTOMS AND COURSE.—In many instances prodromata give warning of the attack. The child is cross and feverish; is hoarse, and coughs with suspicious tone. Such symptoms alone, however, may be of little moment, being quite as indicative of the approach of an insignificant laryngeal catarrh, as of the onset of one of the most fatal disorders of childhood. Even thus early, however, an observant physician may distinguish between the two. In all cases examine the fauces forthwith, although the child do not complain of difficulty in swallowing. Should we find them swollen, and spotted here and there with small, firm, white patches, we have before us the signs of incipient croup, while the same symptom, accompanied by persistent sneezing, and by a profuse flow from the nose, is equally characteristic of laryngeal catarrh. A further diagnostic point is found in the predisposition of the individual. If a child habitually grows hoarse and coughs with a bark upon taking cold, but never exhibits other sign of croup; if his brothers and sisters show no tendency toward the disease, we may feel less concern for him, than for one who already has suffered an attack, or who has lost a brother or sister by this malady. These prodromata may precede the attack itself by one or two days. They are absent, however, in very many cases, the disease setting in suddenly and unexpectedly in all its terrors. Late in the evening, generally, or in the middle of the night, the child is roused from his sleep with a harsh, hoarse, inaudible voice, the deep, soft note of which breaks into shrill, piercing discord, as the swollen vocal chords, already frequently coated by exudation, come for an instant into contact. The cough, which was short and sharp in the beginning, soon becomes harsh, hoarse, and is no longer barking, except when, upon a violent expiratory effort, the air in its exit stretches the chords, and causes them to bulge. At last the cough loses all sound. We see the child cough and speak; we hear nothing.

Besides these symptoms, which are, and indeed must be, entirely identical with those of catarrh of the larynx, and which owe their origin to the thickening and relaxation of the vocal chords, from incipient palsy of the muscles, by which they are stretched, and to the exudation which coats them, there is dyspnœa, a persistent, perilous dyspnœa, characteristic of croup, and rarely seen in catarrh of the larynx, and only then as a transitory symptom. This dyspnœa, which proceeds from narrowing of the glottis, and the occurrence of which, where there is no false membrane, requires further explanation, is peculiar, and not easily confounded with any other form of impeded respiration. In the first place, it is tremendously laborious. The efforts made by the child, in order to draw breath, are very evident. Every muscle, which can aid in expanding the chest, is called into vehement action. He sits up, extends his spinal column, so as more effectually to dilate the thorax by upheaval of the ribs. In spite, however, of every effort, the air can pass but slowly through the contracted rima glottidis. The breathing is remarkably protracted and tedious, and hence, of course, much less frequent than in dyspnœa from other causes (pneumonia, for instance, where the muscles of respiration have no abnormal obstacle to overcome). It also gives rise to an exceedingly characteristic wheezing or sawing sound, which, if once heard, will always be recognized in future.

During these laborious efforts at inspiration, the levatores alæ nasi contract, dilating the nostrils (for, without this instinctive muscular action, the nostrils would tend to close, from the rapid rarefaction of the air within the nose). This "working of the nostrils," however, is not peculiar to the dyspnœa of croup.

Besides this, however, there is another and characteristic sign of croup, which is known even to the laity, and which depends upon the rarefaction of the air, within the thorax, when dilated during stricture of the glottis. We see, namely, that with every inspiration the epigastrium, instead of projecting, is strongly and deeply depressed. When the air within the chest becomes rarefied, the pressure upon the thoracic surface of the diaphragm becoming far lighter than that upon its abdominal surface, it yields, and is forcibly pushed upward, the xyphoid and costal cartilages being likewise drawn in by the inspiration. This, too, is easy of comprehension, if we only bear in mind the mechanism of normal respiration. If the air can enter the air-passages with freedom, the diaphragm, upon contracting, causes its *pars tendinea* to descend, but produces no incurvation of the ribs; for their resisting power is far greater than that encountered by the diaphragm in the elasticity of the lung, or in the feeble pressure of the abdominal viscera.

If, however, the tendinous centre be drawn up by the rarefaction of the air in the lungs, or if it be only fixed and hindered from moving downward, the inspiratory contractions of the muscles of the diaphragm must then, of necessity, cause the arch of the ribs to curve inward. The desire to draw breath, the efforts to do so, and the desperation which its fruitless exertions produce, are evinced in the entire being of the child. Now it begs to be taken out of bed into the arms of its nurse, and from its nurse to be put to bed again. The greatest terror is depicted in its manner; it beats about, throws itself hither and thither, clutches at its throat, pulls at its tongue, as if to remove the obstacle to its breathing. The face is distorted and bedewed with sweat. The look of a child sick of croup is, above all things, sad and piteous.

The circumstance, that children often die of croup, who, during life, evinced signs of the greatest dyspnœa, but in whom, after death, neither pseudo-membrane nor considerable swelling, either of mucous membrane or of the submucous tissue, could be discovered, has given rise to the impression that, in these cases, spasmodic contraction of the laryngeal muscles has constricted the glottis. This view is contradictory to pathological and physiological fact.

In all severe inflammation of mucous or serous membranes, we find not only the submucous, and subserous cellular tissues, but also the muscles covered by the inflamed membrane, infiltrated with serum, sodden, and pale. Even *a priori*, it is not to be supposed that muscles in this condition should be capable of a spasmodic contraction, and *Rokitansky* declares his opinion, from a pathological point of view, that "the infiltrated, pale, relaxed muscular tissue, in croupous inflammation, is stricken with palsy." That muscles in this condition really do lose their contractile power, is shown by the paralytic bulging of the intercostal muscles in pleurisy, and in the loss of peristaltic action of the intestine in peritonitis, or dysentery, from palsy of the intestinal muscles, covered by the inflamed mucous or serous membrane. These, and many other analogous observations, render it highly improbable that the laryngeal muscles should be spasmodically contracted, instead of palsied, where their mucous covering is inflamed. Section of the par-vagum nerve, in young animals (an operation originally practised for an entirely different purpose), furnishes absolute proof, that paralysis of the muscles of the larynx produces dyspnœa; nay, the dyspnœa arising in consequence of this experiment bears so strong a resemblance to croupous dyspnœa, is attended by such similar long-drawn whistling inspiratory efforts, and other signs, that the similarity of the two conditions must strike the most indifferent beholder. But the study of the anatomy of the larynx of a child makes it certain, that a forced effort at inspiration will contract or close the glottis, unless it be held open by muscular action. In childhood, we do not find that triangular space, bounded by the base of the arytenoid cartilage, stretching forward, and inward, to the processus vocales, known as the *pars respiratoria* of *Longet*. In children, the base of the arytenoid cartilage has no extension, the glottis forming a small cleft, running antero-posteriorly, and bounded by the membranous expansion of the vocal chords. These membranes lying obliquely opposed, one to the other, unless the glottis be held open by muscular action, the effect of an energetic inhalation must be to contract, and close the cleft, by rarefying the air within the trachea.

In any juvenile larynx which we may cut out of the body, the glottis is capable of being completely closed by the application of powerful suction to the trachea. As it is of the utmost importance, in the treatment of croup, for us to know whether false membranes occlude the glottis, or whether palsy of the laryngeal muscles, by œdematous infiltration, be the main cause of the dyspnœa, we must carefully note whether inspiration and expiration are in equal degree obstructed, or whether inspiration alone be laborious, and expiration free. In the former, and most common case, false membranes clog the rima glottidis, impeding both exit and entrance of the air; in the latter, crippling of the muscles is the chief cause of the dyspnœa. Upon rarefaction of the air within the trachea during inspiration, the inflowing current, through the nose and mouth, forces the folds of the glottis together; but expiration follows freely, as the air, when expired, drives the vocal chords apart, without any need of muscular aid. Let us bear in mind, too, that the posterior crico-arytenoid muscles, which open the rima glottidis, are more easily paralzyed when the mucous membrane of the pharynx, which covers them, takes part in the inflammation. Thus, it is easily seen why those cases of croup, which the French alone admit to be true croup, cases in which croup-membrane can be seen upon the pharynx, must be by far the most dangerous.

The wide gaping of the glottis during inspiration, when the laryngeal muscles are acting normally, of which I have been able to convince myself as often as I have looked in the laryngoscope, has materially strengthened my conviction of the correctness of my theory, that palsy of the muscles of the glottis forms an important element in the dyspnœa of croup.

It is difficult to say whether any real pain in the larynx forms a symptom of croup. The clutching of the child at its throat may depend upon the desire to remove the impediment to its breathing, which it instinctively perceives. In the beginning of the disease, the expectoration, which is usually scanty, rarely contains masses of shreds, or of coherent false membrane. The pulse, at first, is generally full, hard, and of moderately increased frequence; the face is flushed, and the temperature of the body elevated.

Croup, in a great many cases, exhibits decided remissions in the morning, and through the course of the day, which might almost seem intermissions. (Hence the homœopathists promise that their medicines will not evince their wonderful effect until after a lapse of several hours.) Toward morning the respiration becomes more free. The voice returns. The cough is less frequent; it is hoarse, but not without sound. The fever abates; the general condition appears almost undisturbed; and only the thin piping, or the still suspicious tone of the cough, remains, to recall to mind the scene of terror of the previous night. But beware of building too great hopes upon these remissions. The coming night may bring with it a repetition of the same symptoms, and the greatest danger to the life of the child. The continuance of fever, even if only moderate, and, above all, the presence of pseudo-membrane in the pharynx, should excite the greatest solicitude.

Sometimes the croup exhibits this rhythmical type throughout its entire duration, bad nights following upon tolerable days; in fatal cases, the remissions becoming more and more incomplete, and the nocturnal exacerbations growing more and more formidable. In other instances, which are far more dangerous, the symptoms of croup run a continuous course from the beginning. The remission expected in the morning fails to appear, and death may ensue in the course of two or three days.

When, instead of abating, the malady tends to terminate unfavorably (an event but too common in croup), the scene changes. The flushed face of the child grows pallid, the lips lose their color, the eye, which hitherto has been gazing anxiously about it, assumes a drowsy expression. Quite frequently spontaneous vomiting sets in, while the emetics which we administer remain without effect, and the child grows insensible to sinapisms and other cutaneous stimulants. The respiration becomes diminished, and now the whistling sound of inspiration often ceases; the child lies exhausted in a half-slumber; the symptoms of croup seem gone. It seems to have no more dyspnœa, until, upon awakening from sleep, or after coughing, it involuntarily attempts to draw a long breath. Then the glottis closes; the child, once more in danger of suffocation, springs up, props itself up with its hands, looks desperately around it, anew makes violent efforts to draw breath, and finally sinks back again exhausted, and falls into a state of semi-somnolence. (In young animals in which the par vagum nerves have been cut, we observe precisely these phenomena. Respiration almost free as long as they breathe quickly; respiration impeded in the highest degree the moment they attempt to draw a deep breath—a condition easily understood after the above explanation.)

These changes in the child's condition are a result of gradual blood-poisoning by carbonic acid, overcharge of the blood with this gas forming one of the main sources of danger from this disease.

The above-described train of symptoms is by no means due to engorgement of the brain and its meninges (as has been generally assumed), nor is a child with croup ever cyanotic from impeded respiration alone, excepting when, in the act of coughing, the flow of blood within the jugulars is arrested by compression of the contents of the thorax. A child with croup cannot be otherwise than pale at this stage of the disorder, and the pallor continues until, as palsy of the heart sets in, the contents of the arteries grow less and less, the veins fuller and fuller, and thus a livid tinge is imparted to the pallid lips. As the blood of the veins within the thorax is subjected to a pressure less than that upon the veins without, the tendency of the elastic lung being to contract, and thus to cause the vessels which border upon it to expand; as with each deep inspiratory effort the power of suction of the lung grows stronger (since the draught increases as the lung expands), this suction must reach its highest pitch of intensity; blood will be drawn with greatest power from the external veins into those within the thorax, when any one with constricted glottis rarefies the air within his lungs by trying to draw a long breath. Cyanosis and obstructed evacuation of the cerebral veins can never take place in this way. The process must always have an opposite effect.

When inspiration and expiration meet with equal obstruction, the circulation is somewhat differently affected. As the glottis becomes so much occluded by false membrane, that very little air can enter into, or escape from, the lungs, inspiration and expiration can only be carried on by means of all the auxiliaries at command. Now, as we are able to expel our breath with greater force than we can inhale it, the influence of the forced expiration over the discharge of blood from the thorax outweighs that of the forced inspiration, and then, indeed, cyanosis takes place.

Since the interchange of gases in the lungs depends principally upon the renewal of air contained in the air-vesicles, and as the blood does not give out carbonic acid, and absorb oxygen, unless the air within the vesicles contain less of carbonic acid and more of oxygen than the blood in the plexus of capillaries about it, the necessary consequence of the incomplete respiration in croup, and of the imperfect renovation of the air in the vesicles is, that the carbonic acid which incessantly forms in the blood cannot escape from it into the air of the vesicles which is already overcharged with it. The symptoms described are exactly the same as those produced by the inhalation of carbonic acid. In croup, the carbonic acid created within the body itself poisons the patient, while in the other case the poison is breathed with the atmosphere.

In fatal cases, death almost always takes place with the symptoms
described, through the gradual establishment
consequence of carbonic-acid poisoning. I
of air to the lungs may be suddenly
of a piece of loosened membrane be
by suffocation may ensue.

If the croup take a turn for the be

place gradually with occasional expectoration of quantities of tough sputum, containing a more or less profuse admixture of flakes of coagulum—the cough becoming easier, the voice louder, the symptoms of narcotism disappearing, as the embarrassment of respiration subsides.

In other instances, however, which are far less numerous than is generally supposed, large masses of pseudo-membrane, and often tubular casts of the substance, are thrown out after violent coughing, retching, and vomiting, so that the breathing, till now extremely oppressed, suddenly becomes much more free. The child is safe from immediate danger, if a reproduction of the exudation do not once more occlude the glottis, or a new exacerbation of the inflammation again produce œdema of the laryngeal muscles.

After subsidence of the croupous process in the larynx, when its duration has been somewhat protracted, many children perish from hyperæmia and œdema of the lungs, and of bronchial catarrh. The comparatively ill success of tracheotomy, after protracted croup, is entirely due to these complications, the frequence of which we can easily show to be a necessary result of the previous disease. When the thorax is expanded, and the alveoli are made to dilate without allowing the atmosphere to penetrate into them, the air already contained in the bronchi and air vesicles must be expanded and rarefied. The bronchial mucous membrane and inner wall of the air-vesicles during croup are thus placed in a condition similar to that of a portion of external skin under a cupping-glass. Hyperæmia and increased secretion are the necessary result of the suspension or diminution of the pressure to which the capillaries are habitually subjected. The circumstance recently urged by *Bohn and Gerhardt* in their two valuable works upon croup, that bronchial catarrh invariably and promptly associates itself with croupous laryngitis with constricted glottis, seems to me to argue in favor of the genetic connection of the two processes. With regard to the croupous pneumonia and bronchitis, however, which complicate laryngeal croup in many cases, it is quite otherwise. As I have stated in the opening words of this text-book, it is catarrhal inflammation only which arises in consequence of vascular engorgement of a mucous membrane.* I shall repeatedly recur to the impropriety of regarding other forms of inflammation as an exaggeration or a consequence of simple hyperæmia. "That the danger from croupous laryngitis is considerably heightened by the addition to it of bronchial catarrh" is perfectly admissible; that, however, in real croup, "death always proceeds from bronchitis or broncho-pneumonia" (*Bohn*) is certainly an exaggeration. The symptoms of the secondary croup which complicates measles, small-pox, scarlatina, epidemic diphtheria, and other infectious disorders, will be described hereafter when treating of the diseases themselves.

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* I have no objections to make against the opinions of authors who do not consider catarrh as an inflammation, but rather as derangement of secretion, characterized by swelling and succulence.

Diagnosis.—We have already drawn attention to the points of resemblance between croup and laryngeal catarrh, and have also shown in what respects the two diseases differ. We have merely to add that the pharyngeal patches of croup membrane are of nearly as great diagnostic value as the masses of membranous exudation discharged by coughing or vomiting; that dyspnœa is rare in laryngeal catarrh, and is never persistent, nor is there often much fever, while in croup, fever never fails.

Prognosis.—Children who have passed their seventh year may survive attacks of croupous laryngitis of the utmost intensity, but during the earlier years of life croup is one of the most formidable of all diseases. We have already remarked that the dazzling reports of cures of which many practitioners boast are to be accepted with caution, most of them being based upon an error in diagnosis.

The epidemic appearance of croup undoubtedly renders the bad prognosis of the disease still worse, and though it would be going too far to pronounce croupous laryngitis, complicated by croup of the pharynx, absolutely mortal, yet it is not to be denied that this complication renders our prognosis still more grave.

Among the symptoms, the terror, the restlessness, the full pulse, the flushed face, the hoarseness or aphonia, are of far less prognostic importance than the first indications of commencing blood poisoning. If the face grow pale, the lips colorless, the child drowsy, the sensorium benumbed; if an emetic remain without action; or if, on the other hand, vomiting spontaneously set in, we are rarely justified in expecting a favorable termination of the malady.

Treatment.—Prophylaxis against croupous laryngitis requires the measures already recommended for protection against laryngeal catarrh. Never shut up a child permanently in its chamber because it has once had an attack of croup, nor accustom it to too much clothing. Meanwhile, however, teach the mother not to let herself be deceived by bright sunshine alone, nor to send the child out without paying attention to the direction of the wind. When there is decided predisposition to croup, watch the weather-cock, and keep the child from exposure to a rude northerly or northeasterly wind. It is also advisable to keep the child within doors after sunset. Finally, cold washing of the throat and breast, provided that the skin be afterward carefully dried, is a capital prophylactic where there is predisposition to croup.

As the real causes of croup are obscure, the causal indications cannot, in most instances, be met. Among the laity it is considered a settled fact that the croup is the result of "taking cold." With true fanaticism, the moment that a child becomes hoarse, an incredible quantity of hot sweet milk (which is here preferred to elder-tea) is poured down his throat. Not until the child begins to sweat do they believe him safe, and the foe (often an imaginary one) driven from the field. The teachers of the hydropathic school claim similar results from envelopment of the body in wet cloths, by means of which, "in a great number of cases, they attain the most brilliant success," by thus restoring the repressed action of the skin.

Granting, however, that many cases of croup arise from chilling of the surface, the disease is not so simple a one, the nutritive disorder of the mucous membrane is of far too grave a nature to admit of restoration by the mere production of diaphoresis. In catarrh it may be otherwise. Where hyperæmia alone has sufficed to swell the mucous membrane, active solicitation of the blood to the surface may produce a depletion from the same and cure the complaint.

As, however, it is almost impossible for the laity to distinguish between the two maladies, and as even the physician is often obliged to reserve his decision when first called to see a child suffering from hoarseness, a barking cough, and sudden nocturnal dyspnœa, it is well, in such emergencies, while awaiting the doctor, to give the child hot drinks, to cover him warmly, and to apply a succession of hot, moist sponges to the throat. It has been stated that in many instances, especially of epidemic croup, the inflammation seems to be propagated from the pharyngeal surface into the larynx. If, then, croupous patches be visible upon the fauces, the utmost energy is demanded on the part of the physician. He must not content himself with the application of a few leeches over the throat, as such practice is of very doubtful efficacy. Let him rather remove the false membrane from the tonsils, and thoroughly cauterize the affected part. This treatment is much more to be relied on, and (perhaps from the astringent action of the caustic) is one of the surest of antiphlogistics.

With regard to the management of the disease itself, many physicians, especially country ones, are in the habit of calling for leeches, and emetics, and of forthwith applying one or both articles, if the smallest trace of pseudo-membrane be discoverable. The leeches are to moderate the inflammation; the emetic to remove the exudation. Hardly any one has ever had the courage to treat croup expectantly, and to wait until special incidents in the disease shall call for special measures. It is chiefly to the homœopaths that we owe the discovery, that even a child with the croup may get well without leeches or emetics. Leeches (of which we apply one or two upon the manubrium sterni, or throat of a child under a year old, increasing them in number according to the age) are, moreover, of exceedingly doubtful assistance in croup. In far the greater number of instances they are directly hurtful. Their recommendation is, in great measure, supported upon the erroneous view that hyperæmia and inflammation are identical, hence abstraction of blood will allay inflammation. A really inflammatory process is not interrupted by blood-letting, although it may moderate the collateral hyperæmia in the vicinity of the inflamed spot; however, if a stasis of the blood take place in the mucous membrane of the larynx, if its circulation be interrupted, the blood flows with greater force into the vessels of the neighboring tissues, and produces in them transudation, swelling, and œdema.

We have shown that a part of the danger in croup proceeds from such swelling and infiltration; hence, when we have to deal with a vigorous, blooming child (but only in such a case), we may apply a few leeches to the manubrium sterni. They must never be applied over the larynx, as at that point the bleeding is hard to stanch. In all cases we should apply the leeches ourselves, or employ an expert to do it, who can check hæmorrhage. Among puny, badly-nourished children, leeches are contraindicated. It is most dangerous to exhaust the strength of a child, which he will require at a later stage of the disease to enable him to expectorate with vigor. Blood-letting has no power whatever to prevent the formation of the exudation.

With regard to the employment of emetics, the revulsive action through which they are supposed to exert an influence upon croup is altogether problematic. Still less may we promise ourselves help from their diaphoretic effect. *They are only indicated where obstructing croup-membranes play a part in producing the dyspnœa, and when the child's efforts at coughing are insufficient to expel them.* We have stated, in describing the symptoms, that *impeded expiration should cause us to infer that the glottis is becoming choked by false membrane.* We, therefore, would lay great stress upon this symptom as an indication for emetics. As the formation of pseudo-membrane may take place at a very early period, an emetic, if indicated, may be given early in the disease. In treating croup, preference is given to sulphate of copper over tartar-emetic or ipecacuanha, and, as it seems to me, with reason. Beware, however, of giving this remedy in doses too small, for it may then act with uncertainty, and is much more apt to operate as a poison than when used in full doses. We prescribe ten or fifteen grains of sulphate of copper dissolved in two ounces of water, and let the child take a large teaspoonful of it every five minutes until vomiting sets in. The more complete the remission after the vomiting, the more the membrane thrown out, so much the more reason have we for repeating the emetic, should the peculiar dyspnœa above described recur. If there should be no remission, should no croup-membrane be expelled, or if the expiratory act be free from impediment, the repetition of the emetic is contraindicated. This rule is often broken. How often do we see children incessantly dosed with different salts of copper, even when they have ceased to vomit, and they can do no more service! They lie bathed to the armpits in the bluish fluid which, mixed with curdled milk, flows from their bowels, and in vain turn away their head and push away the spoon containing the repulsive medicine which gives them so much griping and distress.

The application of cold deserves a full trial, in the shape of cold compresses, quickly changed, laid upon the throat of the child, as soon as the signs of croupous laryngitis appear. In families where they are not afraid to use this treatment, we shall have a far more happy result than in houses in which the prejudice against it is not to be overcome. The employment of cold to the skin in inflammation of internal organs, as first recommended by *Kiwisch* in puerperal peritonitis, seems indeed to act as a direct antiphlogistic; and difficult as it may be to comprehend what effect it can have upon an organ separated from it by skin and muscle, experience has here more right to respect than physiological abstract reasoning. (See treatment of pneumonia.) The fanaticism of the hydropaths in this matter, who, while applying cold to an inflamed part, are loath to refrain from other hydropathic measures, has done more harm than good.

We have already recommended touching of the pharyngeal mucous membrane with nitrate of silver as a direct antiphlogistic, and now, after personal experience, cannot sufficiently urge the application of a solution of the lunar caustic to the inflamed mucous membrane of the larynx, as has been much practised in France, and but too little resorted to among ourselves. *Bretonneau*, with whom this local treatment of croup originated, uses a curved rod of whalebone, with a small sponge made fast to its lower end. This is dipped in a concentrated solution of nitrate of silver (ʒ ss to ʒ ij). We press down the tongue of the child, and endeavor to reach the entrance of the glottis with the sponge. There the sponge is immediately compressed by the muscular contraction which takes place, whereby certainly a portion of the liquid, if only a small one, arrives in the larynx.

It is doubtful if calomel have indeed an antiphlogistic, or even a specific effect upon croup, although I cannot deny that very high authority is inclined to credit the beneficial effect of this agent in its treatment; nor that I myself make use of from a quarter to half a grain of calomel every two hours, in most cases of this disease, while my experience leads me to shun the too customary employment of small doses of tartar-emetic and sulphate of copper (tart. stib. gr. i; aquæ, ʒ i—S. ʒ i. 2 h.). (Cupri. sulp. gr. i; aquæ, ℥ i; s. ʒ i. 2 h.) Sulphuret of potassium (℞ potas. sulp. ℈ ss.—℈ j; aquæ; syrup simplic. āā ℥ i M. s. ʒ i. every 2 hours), in spite of the warm eulogium of distinguished authorities, such as *Rilliet* and *Barthez*, has never come much into use, and now is almost obsolete. The proposal of bicarbonate of soda in large doses is obviously based rather upon theoretical grounds than upon actual experience; hence deserves little reliance. The drug has been administered in the hope of loosening the false membrane, and preventing further coagulation of the exudation, or else of improving the state of the blood.

Chlorate of potash, which is a favorite remedy in treatment of diphtheria of the fauces, and of the secondary croup of the larynx, which accompanies it, is also recommended in true primary croup, by those who make no distinction between the latter and the pseudo-membranous laryngitis of acute infectious disorders. I have no personal experience of the action of this article in treating primary laryngeal croup.

When called to the bedside of a child, recently attacked, the physician should not be misled by the idea that, without his immediate and active interference, the child must soon die. Leeches should never be applied, save under the exceptional conditions above stated, and the after-bleeding from the bites should be kept within bounds. It is better, in most cases, to confine one's self to the use of cold, and do not hesitate to put on the first compresses with your own hand, until the parents perceive the relief which they afford. Besides this, if the bowels be confined, administer a clyster, so that the diaphragm may have free room to act. The best is a cold one, of three parts water, and one of vinegar. If the dyspnœa increase, if the respiration be impeded, give an active emetic, without suspending the cold applications. The emetic is to be repeated, under the conditions stated above; if, however, under this treatment, there be no remission, apply a solution of nitrate of silver, at intervals of several hours, to the entrance of the glottis. Do not forget, during the night, that, with the early morning hours, there often comes a remission, nor during the next day, that, in spite of the improvement, the coming night may again bring with it the greatest danger. However well the child may seem to be, it must not quit its bed. The temperature of the room must be kept uniform, and the air rendered moist by means of open vessels of water. Give half a grain of calomel every two hours; aud now change the compresses less often, and cover them with a woollen cloth. Continue meanwhile, but at longer intervals, to use the solution of nitrate of silver. The next night, if the croup grow worse, the same measures are required.

Should this treatment remain without effect, should there be no improvement in the course of ten or twelve hours, do not lose time in giving Schwefelleber, or the "liver of sulphur," so long prized as a specific, carbonate of soda, chlorate of potash, or senega, or other expectorant; but proceed at once to tracheotomy. The earlier we undertake this, so much the more hope may we have that pulmonary hyperæmia, œdema, and bronchial catarrh will not injure our prognosis. Were the results, however, as bad again, it should never be neglected when other means have failed. Even death itself, after performance of the operation, is far less painful, both to patient and relatives, than where it has been omitted.

Besides treating the dyspnœa upon the principles given above, we have also to relieve the paralytic symptoms due to blood-poisoning by carbonic acid. For this purpose, the powerful stimulus obtained by pouring cold water upon the child, while in a warm bath, is of great service. This is also a favorite remedy in treatment of asphyxia by charcoal vapor. Lose no time in making use of it, the moment the child begins to grow drowsy, the skin to cool, the sensorium to be benumbed, or as soon as emetics fail to act; for, at this period, their operation is often of the utmost importance. A few gallons of cold water, poured from a moderate height, over the head, nape, and back of the child, almost always cause it to revive for a while, and to cough vigorously. Thus, sometimes after the bath, masses of exudation are expelled. Other stimulants, such as camphor or musk, are much less effective, and ought not to be employed save when insuperable objections are opposed to the cold effusion. They should be given in large doses, immediately prior to the emetic. (℞. camphor gr. x. Ether acet. ʒ iij. m. S. gtt. x—xv. every quarter of an hour. ℞. moschi. gr. iv. Sacch. alb. ʒ i. m. div. in. ch. vi. s., a powder, every hour or half hour.)

The application of sinapisms to the calves of the legs and soles of feet, repeated bathing of the hands and forearms in water as hot as the child can bear, the use of "flying blisters" to the neck and chest, are recommended, partly to corroborate the action of the stimulants administered internally, and partly as a derivative from the larynx to the skin. Although we do not rate cutaneous irritants very high among the remedies against croup, yet, for want of better or more promising means, we make use of them where the disease is protracted, sometimes improving, and again growing worse; and where we are dissatisfied with the effects of the treatment already described, and yet hesitate to proceed to tracheotomy. In order to accelerate the action of the flying blister, *Bretonneau* advises that the plaster be smeared with a solution of cantharidin in oil, and covered with blotting-paper before application.

## CHAPTER III.

### CATARRHAL ULCERS OF THE LARYNX.

ETIOLOGY.—When the cell-formation, which takes place upon the surface of the mucous membrane in acute and chronic catarrh, encroaches upon the tissue of the mucous membrane itself, producing in it a solution of continuity, a superficial loss of substance occurs, constituting the simple catarrhal ulcer or catarrhal erosion. The pathogeny of this ulcer is easily understood, if we compare it with a very similar process upon the skin. Where a plaster of cantharides has raised the epidermis in a blister, the contents of the blister, in a few days, become turbid from admixture of young cells. These are formed upon the surface of the cutis, through the proliferation of the more deeply-situated epidermic cells. The substance of the cutis is intact. If, however, after opening the blister, we anoint the exposed cutaneous surface with an irritating salve, the cell-formation extends to the substance of the skin, causing its destruction, and forming a superficial sore completely analogous with the catarrhal ulcer of the mucous membrane.

In other cases, the numerous mucous glands which exist in the larynx become the seat of a vast multiplication of cells. They enlarge considerably; their covering is finally perforated, their contents are discharged, and, in place of the gland, there remains a round, crater-formed loss of substance—the second form of catarrhal ulceration, the follicular sore.

Ulcers are rare in acute laryngeal catarrh. In the chronic form, however, especially in the follicular variety, affecting the fauces and larynx of preachers, singers, inveterate smokers, and immoderate drinkers of spirits, there is a decided tendency to ulceration.

This is still greater in the chronic laryngeal catarrh which almost always accompanies pulmonary consumption, independently of tuberculous disease of the larynx. Finally, *Türk* has repeatedly noticed catarrhal ulcers of the larynx, in the vicinity of which scarcely any trace of catarrhal disease could be discovered.

Special exciting causes render certain portions of the laryngeal mucous membrane particularly liable to catarrhal ulceration, namely, the posterior wall of the larynx, the aryepiglottic ligament, the anterior and posterior ends of the vocal chords and the epiglottis, at the point corresponding to the processus-vocalis of the arytenoid cartilage. The places first named are particularly rich in mucous glands, and the tissue of the mucous membrane is loose, as it here contains a lesser quantity of elastic fibre. At the latter-named spots the cause of ulceration seems to be mechanical. In all loud talking the vocal chords are forced toward one another, so that their edges almost touch. When their mucous integument is swollen by catarrh, a constant friction takes place during speaking, which results in excoriation and ulceration. (*Lewin.*)

ANATOMICAL APPEARANCES.—In the beginning catarrhal erosions have either a rounded or an elongated shape, according to the arrangement of the elastic fibres; but they afterward coalesce, forming an extensive loss of substance of irregular contour. The follicular ulcers, however, retain their circular form, even when of long standing, and show less tendency to increase in width than in depth. They readily lead to disease of the cartilage, and, exceptionally, several of them run together, and produce extensive destruction of the mucous membrane, the "catarrhal consumption of the larynx."

The ulcers originating at the anterior and posterior ends of the vocal chords spread lengthways over the greater part of one, or still oftener of both chords. In many cases the loss of substance is so shallow that the chords appear as if they had been only superficially shaved off; in other cases the destruction is more considerable. *Lewin* describes catarrhal ulcers upon the lower surface of the vocal chords, of which, during life, we can only make out the outer border, as a minute fold of mucous membrane, which seems to be inserted under the level of their upper membrane.

In phthisical patients this author has so often found catarrhal ulceration in that portion of the laryngeal mucous membrane where the vocal processes cover the arytenoid cartilages that he describes this laryngoscopic appearance, which hardly ever is met with in persons with healthy lungs, as almost pathognomonic of pulmonary consumption.

SYMPTOMS AND COURSE.—The general symptoms of a chronic laryngeal catarrh are not materially modified when accompanied by ulceration. True, we may suspect the existence of an ulcer when a patient with a harsh, barking cough, of long standing, and chronic hoarseness, running from time to time into aphonia, complains of a sensation of burning, or soreness upon speaking or coughing; but these symptoms (although sometimes so distressing that the sufferer, in order to avoid pain, speaks without moving the vocal chords, that is to say, in a whisper) are often entirely absent, even when very extensive ulceration exists. The addition of painful and difficult deglutition to the other symptoms renders the presence of an ulcer still more probable; and when the epiglottis, the aryepiglottic ligament, or the arytenoid cartilage is involved, this symptom is rarely absent. But as painful deglutition also occurs in a severe case of simple catarrh at this point, no positive inference can be drawn from this symptom alone. Next to the objective signs of ulceration, the admixture of small streaks of blood in the sputa furnishes the most reliable token of its existence.

Among objective signs, the condition of the fauces and gullet is of great diagnostic importance. Experience teaches that follicular ulcers of the larynx are often combined with follicular pharyngeal ulceration. If, then, in patients with long-standing hoarseness and other symptoms of chronic laryngeal catarrh, we find a reddening of the mucous membrane of the soft palate, and see the posterior pharyngeal wall studded with small round, yellowish sores, it is to be presumed that the disease has also invaded the larynx.

The majority of laryngeal ulcers may be brought into view by means of the laryngoscope, especially when situated upon the epiglottis, upon the arytenoid cartilages, on the aryepiglottic folds, and upon the true and false vocal chords.

TREATMENT.—The treatment of catarrhal ulceration of the larynx is almost identical with that of the simple laryngeal catarrh; and, as in catarrh of other mucous membranes, we do not materially modify our treatment where ulceration supervenes upon simple inflammation. It cannot, however, be denied that the cure of catarrhal ulcers of the larynx takes place somewhat more rapidly when the medicaments are applied directly and solely to the sore itself, instead of over the whole mucous surface. Whoever has obtained sufficient laryngoscopic dexterity to enable him to touch the ulcers with lunar caustic in substance, or with a concentrated solution of nitrate of silver, will do well to adopt such local treatment, instead of that recommended in the last chapter, especially instead of inhalation of alum or nitrate of silver in solution. While practising local treatment, however, whether by cautery or inhalation, the dietetic and other internal treatment already described is not to be neglected. The partiality to which specialists are so often prone is not only hurtful to the patient, but injures the credit of new therapeutic measures. When a chronic ulcer of the larynx, which has long resisted a regular course of caustic at the hands of a specialist, recovers under the use of Ems-water and careful nursing of the mucous membrane, perhaps after weeks of absolute enforced silence, it will generally be found that the patient had relied solely upon the local treatment for a cure, and had lived imprudently or absurdly.

## CHAPTER IV.

### TYPHOUS AND VARIOLOUS ULCERS OF THE LARYNX.

ETIOLOGY.—From the teaching of *Rokitansky*, the belief has long prevailed that typhous ulceration of the larynx proceeded from "medullary infiltration of the mucous glands of the larynx with subsequent sloughing," and was therefore quite analogous to the intestinal ulcers of typhus, which are formed by the action of a similar morbid process upon the solitary glands and the glands of *Peyer*.

This mode of origin, however, if it occur at all, is certainly not the sole, nor even the most frequent, source of typhous laryngeal ulceration. *Rokitansky* himself, in his last edition of his pathological anatomy, attributes typhous ulcer of the larynx to diphtheritic infiltration of the mucous membrane; and, indeed, it is entirely in harmony with this view of the matter that typhous ulcers appear upon the most dependent portions of the larynx, in which hyperæmia from gravitation is easily developed, after long-standing disease; and, as is also the case in the lower parts of the lungs, and of the integument of the back and loins, at points most exposed to pressure or mechanical irritation. The most striking observation, however, is that of *Rühle*, according to whom, even in exanthematic typhus (a disease entirely foreign to abdominal typhus, which runs its course without medullary infiltration of the intestinal glands), besides the products of catarrhal, croupous, and the diphtheritic processes, we find ulcers of the larynx exactly similar to those of abdominal typhus.

While the infection of measles is followed by catarrhal, or, in rare cases, by croupous laryngitis, and while the poison of scarlatina does not localize itself in the larynx, excepting by propagation of diphtheritic inflammation from the fauces, the virus of small-pox, in a majority of cases, causes pustular inflammation of the mucous membranes of this organ.

The variolous ulcer has its origin in the propagation of the exanthema from the skin, and from the mucous membrane of the mouth and pharynx. We thus have to do with an eruption of small-pox in the larynx, which, however, as a rule, is complicated by a diffuse croupous inflammation—a secondary croup.

ANATOMICAL APPEARANCES.—The typhous ulcer presents a loss of substance of the mucous membrane, bounded by relaxed discolored edges. Its most common seat is the posterior wall of the larynx above the transverse muscle, and on the lateral edges of the epiglottis. As a rule, it only has a circumference of a few lines. In some cases, however, it extends so as to involve the entire free edge of the epiglottis. In others it is more disposed to penetrate deeply, and thus may lead to laryngeal perichondritis, and to exposure and consequent necrosis of the cartilage.

The variolous ulcer commences by the formation of a soft, flattened, non-umbilicated pustule, which soon bursts, forming a shallow, rounded sore, which readily heals. The croupous exudation which accompanies the small-pox eruption, and which, according to *Rühle*, is often found there when the pustules are wanting, consists of a somewhat thin film, spreading over the swollen mucous membrane. The latter is at first somewhat reddened, but afterward grows paler. After the fall of the false membrane, which usually extends as far as the bifurcation of the trachea, the condition of the mucous membrane is normal, with the exception of a few trifling abrasions.

SYMPTOMS AND COURSE.—Owing to the position which it usually occupies, the typhous ulcer of the larynx does not cause alteration of the voice, unless there be a coexisting swelling, and relaxation of the vocal chords. Pain, or other sensation, is either slight or entirely absent. At all events, the sick, as they lie half slumbering, do not usually complain of it. Hence, we see that, during life, the typhous ulcer is not recognized, nay, cannot be recognized, and is often only discovered by accident upon the dissecting-table. Never neglect, therefore, in typhus subjects, to examine the larynx *post mortem*, even though during life no symptoms of disease of the larynx existed. In other cases the relaxation and swelling of the vocal chords are so great, that the voice becomes rough and hoarse, and in cases where the stupor is not very great, there may even be violent fits of coughing, or of harsh, hoarse, inaudible "hacks." Although these symptoms are not so much signs of typhous ulcer of the larynx as of disease of the mucous membrane causing the ulcers, yet, from the fact of their appearance in the second or third week of the fever, we may diagnosticate the so-called laryngo-typhus from them. Although almost without importance of itself, this laryngeal-typhous ulcer may occasion danger from œdema glottidis and laryngeal perichondritis.

Variolous ulcers necessarily give rise to symptoms identical with those of severe laryngeal catarrh. The two diseases would not be distinguishable, did not the eruption upon the skin and the pustules in the mouth and throat furnish a distinct criterion.

The secondary (variolous) croup, like the genuine, causes hoarseness and aphonia. The cough is generally moderate or entirely wanting. Either because the false membranes are not thick enough materially to occlude the passage of the glottis, or because œdema and palsy of the muscles of the glottis (to which we ascribe a part of the dyspnœa of croup) do not occur here, it is only on rare occasions that dyspnœa, like that of genuine primary croup, is met with in this form of laryngitis.

TREATMENT.—Typhous and variolous ulcers usually heal, with subsidence of the primary disease, and need no particular treatment if uncomplicated by œdema glottidis, or by perichondritis laryngea.

## CHAPTER V.

### SYPHILITIC DISEASE OF THE LARYNX.

ETIOLOGY.—Our knowledge of syphilitic disease of the larynx has been greatly extended and modified by means of laryngoscopy. *Gerhardt* and *Roth* have shown that this class of disorders is much more common than had hitherto been supposed. By means of laryngoscopic examination, they have discovered laryngeal disease in a large number of syphilitic patients who evinced no outward signs of it, and have shown that, besides the grave and destructive disorders already known as tertiary syphilis, the so-called secondary forms—the catarrh, condylomata, and simple ulcer—also occur in the larynx with unexpected frequence. I prefer to base my description of this class of syphilitic affections upon the work of these observers, who state that some of the patients dated their laryngeal affection from a "cold;" and hence think it probable that the localization of syphilis in this organ is, in some degree, determined by fortuitous catarrhal inflammation.

ANATOMICAL APPEARANCES.—The anatomical lesions, arising from syphilitic laryngitis, are often merely those of catarrh, and are quite analogous with those of simple syphilitic angina. Although syphilitic laryngeal catarrh is not distinguishable from other laryngeal catarrhs by any palpable anatomical peculiarity, yet the time of its occurrence, after a primary syphilitic ulcer, its duration, its disappearance upon mercurial treatment, testify as to its specific nature, and to its dependence upon syphilitic infection.

Condylomata and plaques muqueuse are much more frequently observed. They form flattened, reddish projections, and some of them show upon their surface the whitish thickening and loosening of the epithelium, which we see in the condylomata of the pharynx and mouth. The most common situation of condylomata is on the vocal chords, although they also occur at other points, particularly the posterior wall of the larynx, and on the arytenoid cartilages and on the aryepiglottic fold.

Simple (secondary) syphilitic ulcers are, on the whole, rare. No ulcers, accompanying the condylomata in the larynx, existed in any of the cases reported by *Gerhardt* and *Roth.* These authors declare the diagnosis of this form of ulcer to be altogether uncertain, as both the yellow coating upon their base and the luxuriant condition of the neighboring parts are found in other forms of ulcers. Simple syphilitic ulcers occur in most varied positions in the larynx, upon the epiglottis, the true and false chords, or in the lower part of the organ. They are not always, nor even frequently, complicated with ulceration of the fauces.

Finally, there are the well-known extensive and profound tertiary ulcers, which coexist with syphilitic lupus of the skin, and, like the latter, are probably due to the breaking down of syphilitic tubercle. Such ulcers almost always begin upon the epiglottis, which they destroy, more or less completely, not unfrequently spreading thence throughout the entire larynx. As a rule, these ulcers have a dentated, ragged shape, and a smooth base, covered with a yellow coating. They show a tendency to cicatrize at the point first attacked, while the destruction advances at other places. The very voluminous papillary and bulbous growths, which surround the sore, and its deeply-retracted scars, are especially characteristic.

SYMPTOMS AND COURSE.—The simple catarrh and the condylomata of the larynx are among the earliest manifestations of constitutional syphilis which appear. If, then, a person, who, some months previously, has contracted a primary syphilitic ulcer, should begin to complain, without assignable exciting cause, of a feeling of tickling in the throat, should his voice become deep and hoarse, should he acquire a harsh, barking cough, and should these symptoms persist in spite of the most careful management, or should the hoarseness gradually increase to complete aphonia, we may suspect that the symptoms are not dependent upon a simple laryngeal catarrh, but upon syphilitic catarrh, or upon the development of condylomata in the larynx. Thus, it appears, from what we have stated in the previous chapters, about the origin of hoarseness, aphonia, and harsh, barking cough, that both syphilitic and simple catarrhs, condylomata, as well as mucous accumulations upon the chords, are capable of modifying the tone of the voice and of the cough, and of preventing the occurrence of sonorous vibrations of the vocal chords.

The fact, therefore, that condylomata, so situated as not to disturb the vibrations of the chords, do not give rise to hoarseness, needs no further explanation. As, in almost all the cases reported by *Gerhardt* and *Roth*, condylomata of the larynx have been accompanied by condylomata upon other parts, especially upon the mouth and throat, the existence of such growth should awaken our suspicions as to their presence in the larynx, while their non-existence permits us to regard the case as probably one of simple catarrh.

Simple (secondary) syphilitic ulceration seems to belong to a somewhat later period, as its appearance does not coincide with that of simple syphilitic ulceration of the fauces. Its presence should be suspected when, in an individual who, one or two years before, has had primary syphilis, and who has since had secondary symptoms, there arises a disease of the larynx, which neither encroaches upon the cavity of the organ nor exhibits characteristics of other forms of laryngeal disease. Here, too, laryngoscopy affords the surest means of diagnosis.

The extensive and profound (tertiary) ulcerations are the easiest to recognize. They form one of the later links in the chain of syphilitic disorders, and almost exclusively attack patients who have for a series of years suffered first from one form of it, then from another, and have resorted to the various methods of treatment by mercury. The sufferers are here not simply hoarse and voiceless, with harsh cough, with profuse and not unfrequently bloody expectoration, but these symptoms are always combined with a more or less intense dyspnœa. We mark the laborious, long-drawn breathing, so characteristic of stricture of the larynx, with its stridor audible even at a distance. This narrowing of the larynx may gradually become so extreme, from contraction of cicatrices and development of exuberant growths in their vicinity, that respiration becomes insufficient, and poisoning by carbonic acid sets in. In other cases, the dyspnœa suddenly rises to an alarming pitch from the occurrence of œdema of the glottis. The fact that the ulceration spreads gradually into the larynx from the root of the tongue and fauces, and there begins its ravages upon the epiglottis, makes it a duty carefully to examine the region of the larynx of all patients suffering from laryngeal stricture, and to press with the finger upon the epiglottis, in order to ascertain if it have suffered any loss of substance. In fact, the positive or negative result of this examination gives almost certain ground for diagnosis for or against the malady, although a closer insight as to the extent of the process is only to be obtained by means of laryngoscopic examination.

In condylomata and simple catarrh the prognosis is good. It is not so good in the simple ulceration, from which sometimes the grave forms last described seem to develop. In the latter, the prognosis is a very unfavorable one. Most patients die, sooner or later, with symptoms of increasing marasmus, even although the respiration remain sufficient, or be made so by tracheotomy. However, in some cases, a partial improvement at least takes place. Thus, in one far-advanced instance, in which the relatives of the patient were confidently awaiting her speedy dissolution, I have seen an almost complete recovery. In this patient, now a blooming female, there is nothing, save a slight stridor and a deficience in the soft palate, to recall to mind the once terrible malady under which for weeks she lay utterly emaciated, without voice, with racking cough, with profuse and often bloody sputum, and bereft of all hope of improvement.

Treatment.—For the treatment of syphilitic disease of the larynx, the same rules apply which are laid down for the general management of syphilis. In extreme contraction of the orifice, tracheotomy is indicated.

## CHAPTER VI.

### TUBERCULAR ULCERATION OF THE LARYNX.

Etiology.—Laryngeal ulcers, which, until recently, were regarded as depending upon tuberculosis of the larynx, are found in nearly half of all cases of advanced tubercle of the lung. Latterly, we have hesitated to admit any ulcers as tubercular, in which the disease, instead of springing from isolated nodules, develops from diffuse degeneration of the mucous membrane. We doubt the identity of that diffuse interstitial cell-formation, and consecutive necrosis of tissue (infiltrated tubercle), with that discrete neoplastic generation of aggregate masses of conglomerate cells and nuclei (miliary tubercle). We cannot deny, however, that both forms so often occur side by side, that a relationship between the two is highly probable. Until a new nomenclature comes into general use, we shall continue to speak of the one as "tubercular infiltration," the other as "miliary tuberculosis." (See section on "Pulmonary Tuberculosis.")

The circumstance that the affection in question never occurs when the lungs are not the seat of tuberculosis confutes the opinion that it is the consequence of injury which the mucous membrane of the larynx suffers through cough, or through the accumulation of acrid secretion at the affected spot. The cough which generally accompanies bronchial dilatation is often much more violent than the cough of tuberculosis; the secretion of dilated bronchi, or of gangrenous vomica, is more foul, acrid, and corrosive, than that from the cavities formed by tubercle; and yet the ulcers in question never form in cases of bronchial dilatation, or of gangrene of the lungs. We believe it to be beyond doubt, that miliary tubercle of the larynx, as well as the diffuse form, takes root upon the same soil as that from which tuberculosis of the lung is developed.

Anatomical Appearances.—The seat of tuberculosis of the larynx is, in by far the majority of cases, that portion of the mucous membrane which covers the transverse muscles; not unfrequently, however, tuberculous ulcers are found in other places, above all, upon the posterior surface of the epiglottis. Here, but only in rare cases, we may observe gray round nodules of the size of a millet-seed, which afterward become yellow, soften, disintegrate, and leave in their stead round ulcers of the size of a millet or hemp seed (*Rokitansky's* primary tubercular ulcers). Through a fresh deposit of nodules in the vicinity, and through the confluence of several ulcers, an irregularly-shaped loss of substance is finally established, with serrated ragged edges (*Rokitansky's* secondary tubercular ulcers). Much more frequently we notice first a yellowish discoloration of the mucous membrane, which, under the microscope, is found to arise from the infiltration of its tissue with innumerable small cells. The tissues of the mucous membranes gradually become relaxed. First, a shallow, then often a very profound loss of substance, takes place in their tissues. The destruction frequently advances from the posterior wall to the vocal chords, so that their posterior insertions, or even large portions of the chords themselves, are sometimes destroyed.

The epiglottis is not often perforated by tuberculous ulceration of the posterior wall; when it does happen, the contour of the part is still preserved, thus forming a contrast to what occurs in syphilitic ulceration.

Tuberculosis of the larynx is very often combined with ossification of its cartilages. If ulceration reach the cartilages, they become carious and necrosed, so that portions of ossified cartilages are often discharged. In rare instances, the ulceration has perforated the wall of the larynx, producing laryngeal fistulæ and emphysema of the skin.

Symptoms and Course.—When hoarseness supervenes upon symptoms of tuberculosis of the lungs of long standing, we may confidently infer the coexistence of tubercle of the larynx. (There are cases in which the hoarseness of tuberculous patients depends not upon an alteration of texture of the mucous membrane, but upon a paralysis of the muscles of the glottis. To this we shall recur hereafter.) Here, too, hoarseness, at least in most instances, is not the immediate result of a tuberculous ulcer, the latter, as we have seen, being, in the great majority of cases, situated upon the posterior laryngeal wall, and upon the epiglottis. The hoarseness is occasioned by the relaxation and thickening of the vocal chords, and by the secretion which lies upon them. We can thus understand why the hoarseness comes and goes, while the ulcers are always growing and persistent. The mucous membrane of a diseased larynx is more vulnerable than that of a healthy one, and far slighter irritants suffice to produce in it a catarrhal affection.

Nay, just as, without any assignable cause, the parts about every chronic ulcer of the skin become more sensitive, congested, and swollen at one time than at another, so, too, the laryngeal mucous membrane when the seat of ulceration seems always in a state of alternate swelling and detumescence. The nearer the destruction approaches to the vocal chords, so much the more persistent and obstinate does the hoarseness become. If, finally, the ulceration destroys their posterior attachment, it is no longer possible to tighten them, nor to throw them into sonorous vibration. The voice is totally extinguished; speech becomes whispering and inaudible.

In other cases, in which the disease runs a more acute course, symptoms of hyperæsthesia of the mucous membrane are more prominent. It is characterized by great irritability and violent reflex phenomena. The most distressing fits of coughing, brought on by the most insignificant and often inappreciable causes, paroxysms of choking, which not unfrequently end in retching and vomiting, besides hoarseness or inaudible voice; all these very striking and painful symptoms forcing themselves so prominently into notice that the phenomena of tubercle of the lung, if not very far advanced, are thrown into the background. The sufferer declares "that he has nothing the matter with his chest," ridicules the percussion and auscultation, and protests that the only evil with which he believes himself to be afflicted, or which he fears, is the "consumption of the larynx."

It is rare for patients to complain of burning or smarting in the larynx, and usually, too, they are but slightly sensitive to pressure there, even though we push the organ back against the spine. The feeling of crepitation perceptible upon this manipulation is also felt in pressing upon this organ in a healthy person, and is of no diagnostic significance. The expectoration is useless as a means of diagnosis (unless, indeed, pieces of cartilage be ejected), since but a small portion of it springs from the larynx. The shortness of breath, the hectic fever, the night-sweats, the emaciation proceed equally from the coexisting tuberculosis of the lungs. In one case only of pulmonary tubercle, besides the symptoms just described, I have seen intense and gradually-increasing stricture of the larynx. The patient died in a few weeks, after having been materially relieved by tracheotomy. At the autopsy there were found in the larynx the thickening and induration of the submucous tissue (previously described as a cause of chronic stricture), together with tuberculous ulceration.

Examination of the pharynx almost always shows that chronic catarrh exists there also. We find its blood-vessels varicose, and see small vesicles, phlyctænæ, or small, shallow, rounded erosions. The sufferer hawks a great deal; deglutition is difficult. At last it is often impossible for him to enjoy liquid food without choking himself, while solid food passes down more easily. In these cases the closure of the glottis is incomplete.

All of these symptoms, however, will not warrant a diagnosis of tubercle of the larynx unless we are able to show that the lungs, too, are affected. They are all capable of being produced by other kinds of laryngeal degeneration. It is well, therefore, in every chronic affection of this organ, at once to institute an accurate physical examination of the chest, and not to pronounce an opinion until we may have been able to avail ourselves of the revelations of percussion and auscultation. The subjective manifestations often fail us, being frequently obscured by those of the larynx. Hectic fever and emaciation are the only signs capable of rendering the diagnosis almost certain without the aid of physical investigation. By means of the laryngoscope we can easily bring the ulcers on the epiglottis and arytenoid cartilages into view. Of the posterior wall of the larynx above the transverse muscle, we, as a rule, can see at least the upper edge, in form of a fringe, with a few pointed jags of a dirty-whitish color (*Türk*).

The praises of specific remedies in cases of pretended cure of tubercle of the larynx are founded chiefly upon error of diagnosis. On the other hand, a number, although a small one, of actual cures of this malady can be authenticated beyond doubt. Death takes place, in most cases, from exhaustion, or with the symptoms of consumption, which we shall treat upon more fully in discussion of the subject of tubercle of the lungs. In some very rare cases, œdema glottidis is suddenly set up, under which the patient rapidly succumbs.

TREATMENT.—In the treatment of laryngeal tuberculosis, we are not in condition to meet either the indication as to cause or the indication from the disease. The symptomatic indications are, first of all, to combat the burdensome cough and attacks of choking, which not unfrequently rob the sufferer of his rest. The treatment in the main must be the same as that recommended for chronic laryngeal catarrh, small as the result to be looked for may be. The Obersaltzbrünnen and the Emser Krähnchen waters, mixed with equal parts of hot milk, and drunk fasting in the morning, seem, in some degree, to moderate the cough. Do not make any objection to the roe of a herring, to be swallowed fasting, nor to the hope which the patient attaches to this prescription. If the pharynx be reddened; if its blood-vessels be varicose; if phlyctænæ and ulcers be visible in it, swab it with a concentrated solution of nitrate of silver, and let the patient gargle assiduously with alum. In this way we can best guard against the too frequent "hawking," which is in itself a source of annoying cough. The insufflation of lunar caustic, the squeezing of a sponge saturated with a solution of nitrate of silver over the entrance to the glottis, by moderating the cough, sometimes have a palliative effect, if repeatedly applied; and, in the few rare instances in which also pulmonary phthisis recedes, this treatment may even have a radical effect. Here, too, we must concede a certain preference to the direct and exclusive application of nitrate of silver in solution, or substance, to the surface of the ulcer itself, when accomplished by skilful and practised hands.

The most important medicaments in the treatment of tubercle of the larynx are the narcotics. Little as they contribute to the healing of the ulcers, their palliative action upon the burdensome symptoms of the disease is indispensable. It has been customary to prefer the use of hyoscyamus and belladonna to that of opium; nevertheless the preparations of the former remedies are seldom as uniform, and their effects, consequently, seldom are as trustworthy as those of opium.

As a matter of course, the patient whose larynx suffers from excessive irritability from tuberculous ulceration must remain in a uniformly heated and, if possible, in a somewhat moist atmosphere. We forbid him all loud speaking; nay, in especially bad cases, compel absolute silence of weeks' duration. When we reflect that, with every act of speech, the vocal chords are subjected to friction from the air which is driven past them, this direction must seem as rational as in practice it will be found to be serviceable.

## CHAPTER VII.

### GROWTHS IN THE LARYNX.

SOME of the growths which form in the larynx are of the papillary kind, warty cauliflower-like excrescences, produced principally by luxuriant development of the epithelium. Some are true mucous polypi, partial hypertrophies of the mucous membrane rendered prominent by their serous or colloid fluid contents. The mucous polypi are sometimes attached by a pedicle; sometimes they rest upon a base more or less broad; and they vary in size from that of a pin's head to that of a hazelnut. More rarely fibrous tumors are found, and these, when provided with a pedicle, are called fibrous polypi. Scirrhus and medullary cancers of the larynx are found almost exclusively in instances where the malady, having originated in the œsophagus, or areolar tissue of the throat, has spread into it from without. Epithelial cancer appears more frequently in the form of soft, whitish-red excrescences, uniformly covering the mucous membrane, frequently penetrating deeply and destroying the cartilage. In other cases they assume the form of solitary tumors and encroach considerably upon the cavity of the organ. Among the great number of cases of laryngeal tumor which *Lewin* has collected from the literature, twenty-two had their seat upon the epiglottis, nine on the aryepiglottic ligament, twenty-one on the ventriculus morgani; thirty-two on the true, five on the false vocal chords; three on the arytenoid cartilages; eight on the anterior wall of the larynx; while in only two instances were pathological growths observed upon its hinder wall, the most frequent seat of ulceration. *Lewin* seeks to explain this remarkable circumstance by the fact that the latter point is subjected to alternate folding and extension during the motions of the glottis. Such a position would therefore be the more prone to ulceration, while commencing growths would soon break down; so that, instead of tumors, ulcers would form. It is a puzzling fact that tumors of the larynx, and particularly polypi, which, a few years ago, passed for pathological rarities, have lately been observed and described in tolerably large numbers. From the care and thoroughness with which autopsies are conducted at the present day, not only by pathological anatomists and clinical prosectors, but also by the better class of private physicians, it is hardly to be supposed that hitherto most polypi of the larynx have been overlooked in the cadaver. On the other hand, the numerous observations of polypous growths of the larynx come, in great part, from such trustworthy investigators, that we cannot believe that insignificant growths or folds of mucous membrane can have been mistaken for and reported as polypi. Until the introduction of the laryngoscope, a positive diagnosis was impossible, save in rare instances. It is true, that sometimes we could surmise, with a certain degree of confidence, that a tumor was growing in the larynx, when the symptoms of laryngeal stricture began to supervene on those of laryngeal catarrh, and the dyspnœa underwent fluctuations as the varying engorgement or depletion of the growth made it vary in size. The probability became greater, when, in the course of the disease, repeated attacks of suffocation took place, which we could only attribute to contraction or closure of the glottis caused by the sudden change of position of the tumor. However, even the periodical return of such choking-fits, which used to be considered pathognomonic of growths within the larynx, by no means made the diagnosis sure. Certainty was possible in those cases only in which the growth protruded, so as to become accessible to palpation or to inspection, or where the patients coughed up fragments of the tumor.

To-day, the recognition of a tumor in the larynx presents no difficulties; but the majority of the polypi and excrescences so easily and certainly detected by laryngoscopy have not produced the symptoms hitherto described as pathognomonic. Most of the patients had suffered merely from hoarseness, aphonia, or troublesome cough, and many of them had in vain been sent to Obersaltzbrünnen, Ems, or even to Cairo or Algiers, there to recover from their supposed laryngeal catarrh or consumption. It is just this class of cases which shows what high time it is that a greater number of physicians should pay more attention to the laryngoscope, so as not to leave this very important art, so essential for the diagnosis of disease of the larynx, and which is not so very difficult to learn, in the hands of a few specialists. With the aid of the excellent books of *Czermak*, *Türk*, *Bruns*, *Lewin*, *Halbertsma*, and by dint of assiduous practice, the necessary skill may be acquired to enable us, in doubtful cases, to make use of laryngoscopy, to effectually confirm our diagnosis. It is not necessary to examine all patients who are suffering from an acute laryngeal catarrh; and, as the procedure is always a fatiguing one, it would be cruel to subject patients to it who have advanced pulmonary phthisis, with hoarseness and aphonia, and who, in their desolate condition, so often turn to the specialists. If, however, hoarseness, a harsh cough, and other symptoms which we had supposed dependent upon a simple catarrh, persist in spite of sedulous treatment, even though no signs of laryngeal stricture may exist, we ought never to neglect to ascertain positively, by means of the laryngoscope, whether a tumor be not the source of the affection.

In others of the newly-observed cases, however, besides the signs of chronic laryngitis, the other symptoms formerly regarded as pathognomonic were actually present, so that it would have been possible to decide as to the existence of these tumors in the larynx, even before the introduction of laryngoscopy. There was that long-drawn, laborious, stridulous respiration, characteristic of stricture of the larynx, particularly when, after any bodily exertion—mounting stairs, rapid running, and the like—the dyspnœa had increased, and the inspiratory movements become more energetic and frequent. *Czermak* and *Lewin* have called to our attention, that in tumors above the glottis it is frequently inspiration alone which is impeded, while if the growth be below the glottis expiration may be embarrassed.

It finally remains to be told that contrary instances have been met with, which not only evinced no signs of laryngeal stricture, but in which there was neither harsh cough nor hoarseness.

The sole complaint of these patients was, of an ill-defined feeling of distress in the throat, or the sensation as if an accumulation of mucus were sticking in the larynx.

The great variety in the symptoms of laryngeal tumors is easily comprehensible, after what we have taught in the first chapter, about the physiology of the voice. It is only in the cases in which the tumor hinders the approximation of the vocal chords, or interferes with their vibration, that they, of necessity, occasion hoarseness or aphonia. On the other hand, all tumors which do not implicate the functions of the vocal chords cannot possibly give rise to such symptoms. Thus it depends entirely upon the seat of a growth, and upon its size, as to whether it cause the symptoms of laryngeal stricture or not. The treatment of growths of the larynx comes under the domain of surgery.

Since the year 1861, when my colleague *Bruns*, with the aid of the laryngoscope, and without incision, first extirpated a laryngeal polypus from the throat of his brother, the operation, which forms one of the most brilliant advances of modern surgery, has been performed repeatedly, both by *Bruns* and by other surgeons familiar with the use of the laryngoscope.

## CHAPTER VIII.

### ŒDEMA GLOTTIDIS.

Etiology.—During inflammation of a part where the skin is attached to the subjacent region by loose areolar tissue, effusion into the latter often takes place with extraordinary rapidity.

Let us recall to mind the œdema about the eye during inflamed wounds of that neighborhood, the post-horn-like swelling of the penis when œdema besets a chancre of the frænulum. This œdema, called by *Virchow* collateral œdema, is the result of increased lateral pressure, occurring in the capillaries in the region about a focus of inflammation, through the capillary stasis at the inflamed point. The more yielding the tissue is, so much the more does the serum transude.

The mucous membrane of the larynx, at most points, is attached to its cartilages and muscles by a close connective tissue. Upon the epiglottis alone, especially at its root, above all, over the bands which stretch from the epiglottis to the arytenoid cartilages (therefore above the ary-epiglottic ligament), and in lesser degree from these downward, to the superior vocal chords, there is a loose submucous tissue particularly prone to œdematous swelling.

The exciting causes which usually produce the sudden serous transudation into the submucous tissue, known as œdema glottidis, are sometimes acute morbid processes in the larynx, rarely acute catarrh, more frequently the pustulous laryngitis of small-pox, and sometimes chronic diseases, as syphilitic and tuberculous ulceration. Laryngeal perichondritis often gives rise to sudden œdema glottidis, just as an œdema of the prepuce suddenly associates itself to a long-standing chancre.

Finally, in rare cases, a violent angina, an extensive inflammation of the submucous tissue of the throat, or even facial erysipelas, may occasion danger to life, from the supervention of this affection. In all these instances we have to do with collateral œdema, and but very few cases can be added to these, in which œdema glottidis is a partial manifestation of a general dropsy and accompanying Bright's disease, etc. The above-named diseases (with the exception of variola) are more common in adults than in children, and thus it happens that œdema glottidis is observed almost exclusively among grown persons.

Anatomical Appearances.—The serous infiltration which takes place at the points above described is often so great, that the swollen epiglottis projects above the roots of the tongue; and out from its base two great loose pendulous rolls reach backward to the arytenoid cartilages and to the pharynx. They may attain almost the size of a pigeon's egg, and lie so close together, that the entrance of air to the glottis becomes extremely difficult, or even impossible. In rare instances, and, as it would seem, only when one half merely of the larynx is diseased, a single puffy swelling is to be found, which projects inward, and more or less contracts the entrance to the glottis. These swellings are sometimes pale in color, sometimes more or less reddened. If cut into, there flows from the distended meshes of the connective tissue, at times, a clear, serous liquid; at others a turbid, yellowish matter; the swellings collapse, and now the mucous membrane appears to be wrinkled and thrown into folds. A similar evacuation and collapse of the swelling, with folding of the mucous membrane, sometimes occur *post mortem*, when there has been no scarification, and the *post-mortem* appearances correspond but in small degree with the anatomical changes which, a few hours before death, we have had under our finger. In the upper section of the larynx, in which, as we saw, the submucous tissue is lighter and closer knit, the swelling is less visible. We find the mucous membrane itself covered with flakes, or plastic layers, the laryngeal muscles pale, discolored, and sodden.

SYMPTOMS AND COURSE.—Œdema glottidis commences with the usual symptoms of an acute or chronic laryngeal ulcer. There is a rapidly-increasing hoarseness, which soon passes over into aphonia, and a harsh, barking cough (symptoms which prove that the vocal chords, too, are swelled, or that the infiltrated laryngeal muscles are not in condition to render the chords tense). But, simultaneously with these symptoms, there arises the most frightful dyspnœa. As soon as the air in the trachea becomes rarefied, the swellings described above settle themselves over the upper aperture of the larynx, and the laborious, long-drawn, whistling respiration can bring but little air into the lungs. This inspiration, audible at a distance, and only practicable with body bent forward, arms planted, and by coöperation of all the auxiliary muscles of respiration, is followed by an expiratory movement, which is almost free, although sometimes noisy; for the exit of the air drives asunder the obstructing swellings just as the inspiration sucks them together. *Pitha* describes the inspiration as "protracted, forced, sharp, resonant, whizzing, and hissing; the expiration as short, easy, inaudible, scarcely perceptible."

The symptoms of œdema glottidis, then, are very similar to those which we have described as belonging to croup, a similarity which, after a physiological explanation of the symptoms, is seen to be a natural consequence. Nevertheless, we shall seldom mistake the two diseases, when we bear in mind that croup occurs almost exclusively during childhood—œdema glottidis almost as exclusively among adults; that croup almost always attacks individuals previously in good health, œdema glottidis scarcely any, save such as have already suffered from acute or chronic disease of the larynx. Moreover, the distinction between the two diseases is facilitated by the great disproportion between inspiration and expiration in œdema glottidis, which, in croup, is neither so pronounced nor of so long duration. Finally we may, in many cases, succeed in seeing the swollen epiglottis as a reddened, pear-shaped tumor behind the root of the tongue, and in almost every instance, if we introduce the finger with sufficient boldness, we may manage to feel the two swellings.

The dyspnœa, which, in severe cases, increases from minute to minute, is usually associated with a feeling of a foreign body or other impediment in the throat. "Here it lodges," "here—in the throat"—"tighter and tighter." "It is strangling me." "I can't stand it!" "I'm choking!" these are the words which, according to the classical picture of *Pitha*, the frightfully-terrified sufferers gasp out with tremulous haste and gesture. Fear and desperation are depicted in their entire being; they dash themselves about, they spring up, groan and sob—until gradually the countenance grows livid and lead-colored, the extremities cool, the pulse small and irregular, the sensorium benumbed. Then the patient falls into a stupor, rattlings in the chest begin to be heard, and death sets in with the symptoms of œdema of the lungs. These final manifestations, also, are the same which we studied in croup, and depend not upon impeded evacuation of the cerebral veins, but upon poisoning of the blood with carbonic acid (see above).

TREATMENT.—Blood-letting, leeches in large numbers to the throat, blisters to the nape of the neck, emetics, drastic cathartics, hot foot-baths, are the customary prescriptions which are usually applied, *pêle-mêle*, as soon as this frightful malady has declared itself. We cannot allay œdema of the prepuce by such measures, and they generally are applied in vain in œdema glottidis. Only when the danger is not urgent may we diminish the mass of blood by efficient blood-letting, and by giving half a drop of croton-oil hourly, in order to decrease the volume of the blood in the vessels, through copious transudation of serum into the intestine. Experience teaches that, after great hæmorrhage, and after inspissation of the blood, through profuse loss of its water, the vessels greedily take up liquid from the organs, and that, during cholera, even large pathological effusions have thus been absorbed. There is, therefore, some theoretical support for such treatment, although little success can be ascribed to it in practice. As but little air passes the larynx, in spite of the most forcible inspiratory efforts, that which the bronchi already contains becomes rarified, and, just as in the skin upon which a cupping-glass has been applied, so upon the mucous membrane intense hyperæmia arises, usually accompanied by increased secretion. The dyspnœa is greatly augmented by this collection of bronchial secretion, so that loud moist *râles* become audible. In cases like this, but only in such cases, an emetic is indicated, and is often productive of the best results. It is sometimes necessary to repeat it.

Local treatment is of far more value. The effect of slowly swallowing small bits of ice is sometimes of remarkable benefit. Under this treatment I once witnessed the recovery of one of my colleagues, in whom suffocation seemed so imminent that we hardly dared to defer tracheotomy. The insufflation of pulverized nitrate of silver, or its application in solution, is of questionable service. When the ice fails, we should endeavor to effect scarification of the swelling, either by means of the finger-nail or with a bistouri, guarded with adhesive plaster almost to the point. If we should be unsuccessful, or should the scarification be without effect, or should the symptoms of carbonic-acid poisoning arise, the pulse growing small and irregular, and the senses benumbed, we must proceed forthwith to tracheotomy and insert a canula until the danger is past. In cases like this, the operation, as a whole, is more successful than in croup, and life has been preserved for months in cases where œdema glottidis has occurred in tuberculosis of the larynx.

## CHAPTER IX.

### LARYNGEAL PERICHONDRITIS.

Etiology.—The perichondrium or fibrous tissue immediately in contact with the cartilages is tolerably firm and resisting in texture, so that it long withstands any ulcerative process which advances from the mucous membranes. When finally perforated, the cartilage is laid bare, and its connection with its nutrient vessels is suspended. The portion thus denuded, and deprived of nourishment, sloughs off, and is discharged. We have already stated that these necrosed bits of cartilage generally show traces of ossification, which is one of the early effects of ulceration of the mucous membranes of the larynx.

By perichondritis laryngea, however, we do not generally mean inflammation and ulceration of portions of the perichondrium, penetrating from without inward, but refer rather to an affection in which an exudation forms between the cartilage and the perichondrium, and by which the latter, from its density and impenetrability, undergoes very extensive separation. Necrosis of the cartilage is the natural result of so great a detachment from its nutrient vessels.

Its exciting causes are sometimes ulceration of the laryngeal mucous membrane above mentioned, in which, however, the perichondrium, instead of suffering penetration from without, becomes the seat of suppurative inflammation, which causes an effusion between perichondrium and cartilage.

In other cases the malady arises independently, or at least, without previous inflammation of the mucous membrane, and chiefly in persons with constitutions broken down by syphilis, mercury, or by the infection of typhus, septichæmia, and the like; and sometimes even in subjects who apparently are robust. Of course, in the latter instance, "catching cold" is assigned as the cause; the inflammation is called a rheumatic inflammation, and the destruction of the larynx which follows is called a rheumatic laryngeal phthisis.

ANATOMICAL APPEARANCES.—The point of preference of this disorder is the perichondrium of the cricoid cartilage; but from this point it spreads rapidly to the coverings of the other cartilages. At first there is only a small abscess between the latter and its sheath. Very soon, however, the cartilages are floating in a sack of pus, formed by the perichondrium. They become rough, degenerate, discolored, afterward thinned and softened, and then, not unfrequently, fall to pieces, fragment by fragment. At last, the pus bursts through the perichondrium, and runs into the submucous tissue of the larynx. At times, too, the mucous membrane is perforated; pus and fragments of cartilage fall into the larynx and are coughed up; or else the pus forces its way to the external surface. Abscesses and sinuses of the neck are thus formed, and the pus and fragments of cartilage are discharged externally or into the pharynx. In rare instances recovery has taken place, the lost cartilage being replaced by dense fibrous tissue.

SYMPTOMS AND COURSE.—As a rule, the symptoms of a disease are not rendered easier of comprehension by any arbitrary and artificial classification according to stages, but rather the reverse. When, however, as in perichondritis laryngea, very distinct phases form natural divisions in its course, the study by stages is both commendable and practical.

In its first stage the symptoms are obscure, but, as in all inflammation of dense unyielding structures, the disease is attended by greater pain than occurs in other affections of the larynx; and, as the inflammation generally begins at an insignificant point, the painful region is also quite limited in extent. One might readily be led to suppose that a foreign body were lodged in the larynx; the more so, as the pain is combined with an irrepressible cough.

In the second stage, hoarseness, aphonia, harsh cough, and characteristic symptoms of intense dyspnœa and laryngeal stricture set in, arising either gradually, as the perichondrium becomes more and more distended by the increasing volume of pus, and is pushed further into the cavity of the larynx, or suddenly, by its perforation and by effusion of the matter below it, into the submucous tissue. Many patients perish in this stage.

In some cases a third stage is added. The pus which has accumulated under the perichondrium, or the submucous tissue, bursts through its confines. The symptoms of laryngeal stricture disappear. I have seen a young girl in the most frightful danger of suffocation relieved on the instant, and rendered completely convalescent, after expectorating quantities of pus, and with it the entire left arytenoid cartilage, in a state of maceration.

But, even in such cases, after a time, the patients usually perish in a state of marasmus, in consequence of the continuance of the malady, the suppuration, and the fever by which it is attended.

TREATMENT.—The treatment of laryngeal perichondritis can, of course, be only a treatment of symptoms, seeing that the disease is not generally recognizable until after the effusion has formed in the submucous tissue. There then remains nothing to do except to perform tracheotomy, and this operation and the opening of any abscesses about the neck are about the only measures at our disposal, and even they are merely palliative.

### *NEUROSES.—NERVOUS AFFECTIONS OF THE LARYNX.*

Deranged sensibility of the larynx, excessively exalted excitability of its sensory fibres (hyperæsthesia), and their abnormally diminished sensibility (anæsthesia) are never observed as independent diseases. We may count certain cases of globus hystericus and of spasmodic cough, in hysterical persons, to the first of these forms. True, persons suffering from the latter complaint do not complain of abnormal sensibility in the larynx, but the coughing-fits to which they are subject are to be regarded as reflex phenomena, independent of the morbid condition of the sensory nerve-fibres, whose excitability is increased. The neuroses of the motor function in the larynx are divisible into *hypercinesis* and *acinesis;* into spasm and palsy. We shall treat of each in the following chapters.

## CHAPTER X.

### SPASM OF THE MUSCLES OF THE GLOTTIS—SPASMUS GLOTTIDIS—ASTHMA LARYNGEUM—ASTHMA ACUTUM MILLARI—ASTHMA THYMICUM—LARYNGISMUS STRIDULUS.

ETIOLOGY.—This disease depends upon a morbid excitement of the nerves, by means of which contraction of the muscles of the glottis is effected. By uniform shortening of all the muscles at once, the vocal chords become tightly stretched, and the glottis is closed. The term laryngeal asthma, or laryngismus stridulus, however, is not to be applied to the occurrence of such reflex symptoms during any inflammatory disorder of the laryngeal mucous membrane. It seems rather to be an independent affection of the par vagum, or of its recurrent branch, due either to pressure along some part of the course of one of these nerves or to centric irritation at the root of the vagus; or else we may be compelled to regard its exalted sensibility as a reflex phenomenon arising from excitement of some other nervous trunk. In most cases the pathogeny of this disease is obscure.

Spasm of the glottis occurs almost exclusively during childhood, and especially in the first year of life. It is most frequent during the period of the first dentition. *Romberg* considers congenital predisposition to exist beyond doubt, as in many families almost all the children in succession are affected by the disease. In great cities, and among children brought up by the bottle, it seems to be more common than in the country and among children who receive the breast. Among adults, none but hysterical persons suffer from spasm of the glottis, and these only exceptionally. There have been solitary cases noted in which hysteria has produced death by protracted spasmodic closure of the glottis, but I have seldom seen instances in which such spasm had attained even a dangerous degree of intensity in hysterical persons.

Anatomical Appearances.—Enlargement of the thymus gland, which *Kopp* regarded as the organic cause of spasm of the glottis, in many instances does not exist. This is also the case with the soft occiput of *Elsässer* (a rachitic phenomenon). Enlargement and degeneration of the glands of the throat and bronchi, the occurrence of hypertrophy, of hyperæmia, or of exudative processes in the brain, are in part accidental, and in part (as we have said regarding the pathogeny) are matters not always found *post mortem* after asthma laryngeum. At all events, we must find the larynx apparently sound if the autopsy is to bear out the diagnosis.

Symptoms and Course.—The course of the disease, like that of most neuroses, is an interrupted one, marked by paroxysms and intervals of exemption. A sudden violent interruption of the respiration, which may last for several minutes, is pathognomonic of the seizure. By-and-by the air again begins to penetrate into or out from the glottis, which at first is completely closed, and still remains contracted. In addition to this, the attack is accompanied by the often-mentioned prolonged whistling, inspiratory noise, the fear, the restlessness, the livid countenance, the strenuous contraction of the inspiratory muscles, and the position upright, or bent forward. After a few minutes, as soon as the child has forgotten his fright, he is completely restored. There is no cough in laryngeal asthma, as the laryngeal mucous membrane and the vocal chords are healthy; nor, as the vocal chords are neither relaxed nor thickened, is there any hoarseness. If, therefore, we only hold fast to the idea that laryngeal asthma is a nervous affection of the par vagum, it becomes easy to avoid all confusion of it with croup, or with those nocturnal attacks of dyspnœa which accompany catarrh of the larynx.

In many instances the spasm does not confine itself to the motor fibres of the vagus alone. Sometimes spasmodic contractions of the fingers and toes, or of the hands and feet, accompany the attack, or cramps of these parts alternate with spasm of the glottis. Occasionally even general convulsions occur, in which the sufferer may perish.

The paroxysms thus described take place at varying intervals; a week or more may pass without their repetition. In bad cases the fits multiply, and follow one another more closely, and it is these cases that are most apt to be accompanied by general convulsions. There always remains a great tendency to relapse, which is still to be feared, even though a child have remained for months without a paroxysm. Finally, instances have no doubt been observed in which laryngeal asthma has shown itself but once, never again to recur. In rare instances a paroxysm terminates in suffocation, the closure of the glottis, and consequent privation of oxygen, outlasting the endurance of the organism. The pallid face assumes a deathly hue, the muscles are relaxed, the child sinks back and expires.

TREATMENT.—The indication as to cause cannot be met, as the causes of spasm of the glottis are obscure. However, we should endeavor most carefully to allay all disorders of digestion and nutrition in children who show a tendency to this malady, before we proceed to the employment of specifics of doubtful efficacy. This is what is meant when we prescribe calomel, rhubarb, and other remedies in spasm of the glottis. Children fed by hand should be placed to the breast when they show signs of the disease. In older children, examine the milk, and cause the nourishment in use at the time of the attack to be changed, and so forth.

In hysterical glottic spasm, the causal indication first of all demands treatment of the main disease. Meantime the hysterical symptoms, like all other hysterical manifestations, are to be combated by psychical measures. I have cured both glottic cramp and glottic palsy by means of local faradization of the laryngeal muscles, the treatment undoubtedly acting solely by the psychological effect which it produced.

The indicatio morbi in this obscure and incomprehensible affection is equally difficult of fulfilment. *Romberg* recommends and lauds the effects of the aqua fœtida anti-hysterica which he orders for children in their first year, mixed with equal parts of simple syrup, a small spoonful four to six times a day. If, in spite of the assafœtida, the seizures recur, we may give musk instead, a medicine almost universally prized as a specific. (Moschi gr. iij.—iv., gum. mimos. ʒ ss., syr. simpl., aqua fœniculi, āā ℥ j, liq. ammon. succin. ℈ j. ℳ. s. a teaspoonful every two hours.)

During the fit it is impossible to give the patient medicine, as he is unable to swallow. We should instruct the parents to take the child up as soon as the paroxysm begins, to fan it in fresh air, to rub its back, and to administer a clyster of camomile or valerian tea. It is well to have a mustard plaster in readiness, and to lay it upon the precordium when the fit occurs. If the injections of camomile or valerian fail, it is advisable to substitute a clyster of assafœtida. (Assafœtid. ʒ ss—i, vitell. ov. no. j. ♏ f. emulsio. c. infus. valerian ℥ ss—℥ iv. S. for two clysters.)

## CHAPTER XI.

### PARALYSIS OF THE MUSCLES OF THE GLOTTIS.—DYSPHONIA (APHONIA PARALYTICA).

It is not improbable that, in many cases of supposed spasm of the glottic muscles, instead of spasm, there has been palsy. According to *Romberg*, the signs of palsy of the recurrent laryngeal branch of the par vagum consist in fits of oppression, amounting almost to suffocation, in noisy, hissing respiration, and whispering, hoarse voice, and, above all, in dyspnœa, aggravated by every vigorous inspiratory act, symptoms easily accounted for, at least in children. In treating of the dyspnœa of croup, we have alluded to the strong resemblance between it and that produced in young animals by dividing the par vagum or its recurrent branch; and we then gave so full an account of the physiology of this fact that we may now refer the reader to what has already been said. Turning from this rare and insufficiently-studied form of paralysis of the glottis, let us now briefly direct our attention to that form of loss of power of its muscles in which the respiration proceeds undisturbed, while the defective innervation of the muscles of the larynx modifies the vibrations of the vocal chords, or renders the occurrence of sonorous vibrations impossible. We are accustomed to call these cases "nervous," or, better, paralytic dysphonia, or aphonia, or phonetic paralysis.

Etiology.—The fact of the existence of a paralytic aphonia was no doubtful matter, even before the introduction of laryngoscopy. Instances in which loss of voice constituted the sole symptom of laryngeal disease, which had arisen suddenly, and afterward as suddenly had subsided, scarcely admitted of any other interpretation than this. But it has only been by means of the laryngoscope that we have obtained more accurate knowledge of the important part which the motor disorders of the laryngeal muscles play as the proximate causes of hoarseness and aphonia.

In most cases in which laryngoscopy has discovered imperfection, or total inaction of one or of several muscles of the larynx, the palsy was one of the symptoms of acute or chronic catarrh, or other laryngeal affection. This form of paralysis is very closely related to the subparalytic condition which we at times observe in the corresponding parts of the bronchi, stomach, intestines, and, above all, of the urinary bladder when in a state of chronic catarrh. Indeed, there is hardly any doubt but that the muscles, or their finer nervous fibrillæ, participate in the nutritive disorder of the mucous membrane. In other instances the paralysis manifestly depends upon disturbances of nutrition in the course of the par vagum or its ascending branch. To this category belong the interesting cases of phonic palsy resulting from pressure and tension of the recurrent branch of the par vagum by aortic aneurism, carcinomata, enlarged or degenerated bronchial glands, pleuritic thickenings about the apices of tuberculous lungs, etc. Certain observations render it quite probable that "a cold" may produce nutritive disorder in the vagus or the recurrent, whereby its irritability is destroyed, so that the muscles which it supplies become paralyzed. Hereafter we shall learn that rheumatic palsy of the facial, a cutaneous nerve much exposed to the influence of cold, is one of the most common instances of this form of paralysis. It is doubtful whether palsy of the glottis arising from metallic poisoning, especially lead-poisoning, ought to be regarded as of peripheral or centric origin. This holds true also in the cases of the paralytic aphonia which often remains after typhus and diphtheria, and in the still more enigmatical malarial palsies.

It is very rare for a phonic palsy of the glottic muscles to have a centric origin—that is, for it to arise from an affection of the brain, or of the cervical portion of the spinal cord. The majority of cases of serious disease of the brain, in which the sufferers lose the power of speech, are not taken into consideration here, as in them palsy of the glottic muscles can neither be proved, nor is it even probable—the few shambling and awkward words which this kind of patient is still able to utter being usually pronounced in a loud, unaltered voice.

Next in frequence to the forms just treated of comes hysterical aphonia. I have met with this affection with comparative frequence, both in males and females. We cannot properly include hysteric palsy either in the centric or peripheral forms of the disease, in the ordinary meaning of the term. I prefer to follow the example of *Romberg*, who calls certain forms of spasm "psychical spasm," and to designate hysteric palsy as "psychical palsy." In this affection the excitability of the peripheral nerves is manifestly retained, and its dependence upon nutritive derangement in the motor centres is in the highest degree improbable. On the other hand, psychological influences have so marked an effect upon the appearance and disappearance of hysterical palsy, and especially of hysterical aphonia, that we may almost with certainty ascribe its origin to that portion of the brain concerned in the function of the higher psychical acts.

ANATOMICAL APPEARANCES.—The anatomical alterations which give rise to phonic palsy of the glottis are but rarely susceptible of demonstration in the cadaver, excepting in cases where the paralysis is dependent upon structural change in the larynx, or in which the nervous trunks are compressed by tumors, pleuritic induration, and the like. *Türk* and *Gerhardt* have shown that, in cases of long-standing palsy, the laryngeal muscles, like other muscles, undergo atrophy and fatty degeneration.

SYMPTOMS AND COURSE.—We are not well enough acquainted with the physiological action of the muscles of the glottis, and, moreover, our knowledge as to how far lack of power in one can be compensated for by additional exertion on the part of others, is insufficient to enable us to say with confidence what modification of the voice will naturally follow palsy of any individual muscle, or group of muscles. Pathological observations of pure paralysis of the glottis, independent of relaxation of the mucous membrane, are not numerous enough as yet to admit of a clear, comprehensive view of the relations of its partial paralysis to any particular derangement of phonation. Meanwhile, through the labors of *Türk* and *Lewin*, and, above all, through that of *Gerhardt*, the path is already beaten out, and some ground has been gained. Consistently with the purpose of this volume, I must confine myself to calling attention to a few, merely, of the most important of the facts set forth in these detailed works.

As palsy of the muscles of the glottis generally prevents the normal tension, and approach to one another of the vocal chords, hoarseness and aphonia are the most common symptoms of this affection, but in the majority of cases the approximation and tension of the vocal chords in straining, coughing, and swallowing are not impeded. This form *Türk* calls "phonic palsy of the glottis-closers," in contradistinction to the rarer "general palsy of the glottis-closers," in which the performance of the acts just alluded to is also embarrassed by the non-closure of the glottis. But hoarseness and aphonia are not the only forms of dysphonia in the disease in question, since the palsy does not always affect the glottis-closers. Cases occur, not only as a result of catarrh, but also of compression or irritation of the recurrent, in which the muscles of one half of the larynx become paralyzed, the other half remaining unaffected, and in these cases it sometimes happens that a permanent falsetto voice is the result, a circumstance which is to be explained by the supposition that the sound vocal chord vibrates in normal manner, while the edge of the palsied chord remaining near the median line, but subjected to little or no tension, cannot be thrown into vibration. Palsy confined to the thyro-arytenoid muscles, by which merely the shortening and tension of the vocal chords are embarrassed, according to *Gerhardt*, results not only in the production of too deep a tone, but also in incapacity to produce a tone other than this deep one; hence a true monotony of the voice. These brief hints may serve as incentives to study of the works mentioned, and induce new investigation of this interesting subject.

The diagnosis of palsy of the glottis is of course only possible by means of the laryngoscope. In palsy of the glottis-closers, laryngoscopic observation reveals that, during attempted phonation, the rima glottidis remains open, and the chords do not tremble as they should do. In palsy of one side of the glottis, the point of the arytenoid cartilage stands out in a remarkable manner, reaching to the median line, or even overstepping it toward the other side. The inner edge of the palsied chord comes almost to the middle line. Upon drawing breath, or upon an attempt to speak or cough, there is little or no motion in the arytenoid cartilage and the vocal chord of the affected side. In palsy of the thyro-arytenoid muscle, according to *Gerhardt*, the glottis vocalis forms a somewhat wide ellipse when the patient attempts to speak.

TREATMENT.—In treatment of palsy of the glottis, it is important above all things to meet the causal indication. When the palsy has arisen in the course of a catarrhal attack, energetic local treatment of the main affection, particularly by use of solution of nitrate of silver, promises the best results. In paralysis attributable to derangements in the course of the nervous trunks, a treatment of the cause is in many instances impracticable; in others, the reabsorption of a strumous deposit, the resolution of an enlarged lymphatic, judicious management of metallic poisoning, perhaps the administration of quinine, by allaying the cause, cure the palsy. In centric paralysis, therapeutics are always powerless. On the other hand, in hysterical cases, a systematic and cautious management of the primary complaint most frequently has a permanent effect.

We can seldom meet the indications of the disease itself, as most of them proceed from irreparable nutritive disturbance of centric or peripheral nerves. An exception is made in this respect by the somewhat small class of cases in which the normal irritability of peripheral nerves has suffered from too long-continued rest, or in which it has been imperfectly reëstablished, after removal of the original cause of the palsy. In methodical excitement of the nerves by the induced electric current, we possess a most efficacious remedy. The indication for the employment of localized faradization having grown more precise, the curative power originally ascribed to it, which upon its introduction into practice was estimated to be a panacea against all forms of palsy, has, within the past few years, grown much more limited, exactly as was predicted by *Ziemssen*, who distinguished himself at the time of its introduction.

It is therefore a most remarkable circumstance that localized faradization should seem to have a curative action, beyond all comparison greater in palsy of the glottis than in all other forms of paralysis. In the few years which have passed since we have learned to diagnosticate glottic palsies with certainty, an astonishingly great number of cures have been brought about by the application of the induced current. (*Althaus* alone has cured eleven out of thirteen cases.) In certain instances the sufferers were made well of their aphonia at the first sitting. In the majority of cases an improvement, at least, was apparent at the first application, and the treatment remained without effect in but a comparatively small number of those upon whom it was employed. Had these cures been performed upon cases of mere hysteric aphonia, they would be easy of explanation, since, according to my previous experience, I have often enough seen hysteric aphony made to vanish by means of psychical emotions of a lighter kind than that which the application of the electrodes to the throat produces; but these cures were performed in a great variety of forms of glottic palsy. From my great reliance in the trustworthiness of the witnesses, I do not feel the slightest doubt as to the facts; but I hope that further observation will furnish an explanation to this very remarkable phenomenon.

In order to stimulate the laryngeus superior, according to *Gerhardt*, we select the point of the throat corresponding to the superior horn of the thyroid cartilage; to excite the recurrent, we choose the point corresponding to the thyroid cartilage itself. To faradize the recurrent, it is best to apply one electrode at the spot above named, and the other over the manubrium sterni.

# SECTION II.

## *DISEASES OF THE TRACHEA AND BRONCHI.*

## CHAPTER I.

### HYPERÆMIA AND CATARRH OF THE AIR-PASSAGES AND BRONCHIAL MUCOUS MEMBRANE.

ETIOLOGY.—We have already stated that every considerable hyperæmia gives rise to a series of nutritive and functional disorders, known as catarrh; and that, if catarrh be included among the inflammatory affections, the terms hyperæmia and inflammation must be regarded as synonymous, although the words are not generally considered identical in meaning.

Predisposition to catarrh of the bronchial mucous membrane is as variable as is predisposition to catarrh of the larynx, developing either readily, or with difficulty, from the same causes, according to individual peculiarity. "An increased susceptibility of the skin to change of temperature," or an "augmented vulnerability of the mucous membranes," is simply an hypothetical assumption to which we have recourse, where no other explanation is at hand, to account for the excessive liability in certain persons to suffer from catarrh of the bronchi upon slight exposure. Experience, however, permits us to bring forward certain particular conditions as predisposing causes. In the first place, in childhood, and especially during the period of dentition, there is a strong tendency to catarrh of the mucous membrane in general and of the bronchi in particular. We daily hear it said that children have "a tooth-cough," and hear intestinal catarrh of this period called a "tooth-diarrhœa."

In middle age this predisposition diminishes; in old age, again, it is more marked, senile chronic catarrh becoming a most frequent complaint, and furnishing a large contingent to the hospitals and infirmaries. Secondly: badly-fed, flabby individuals show unmistakable general predisposition to catarrh, and, when exposed to the action of even trifling irritants, are much more liable to bronchial catarrh in particular than well-nourished persons of firmer fibre. This predisposition depends, in part, probably, upon increased susceptibility, or feebler capacity for withstanding noxious influences; but it may also in part be attributed to the poor nutritive condition and weak resisting power of the walls of the capillaries, and to the softness and yielding nature of the tissues through which they run. At all events, an augmented tendency to hyperæmia and to increased transudation would result from such a condition. The disposition to catarrh in general, and hence to bronchial catarrh, which we observe in scrofula and rickets, should also be placed under this heading. Thirdly: those who have often previously suffered from bronchial catarrh show an increased proneness to the disease; chronic disease of the parenchyma of the lungs augments this inclination, if it does not actually give rise to the disease itself. The old saying, "*Ubi irritatio ibi affluxus*," still holds good; although the afflux is only produced by dilatation of the vessels leading to the point of irritation, or by defective resisting power of the vascular walls, and must not be supposed in the least to be due to attraction. Lastly: it must be mentioned that effeminacy seems to augment the liability to this affection, which is less among those who inure themselves to exposure.

The exciting causes of bronchial catarrh, which act more or less readily, according to the tendency of the individual, are as follows: First: it may proceed from an impeded evacuation of the bronchial veins. Let us not forget that the bronchial arteries which spring from the aorta, or intercostal arteries, transmit only a portion of their blood into the bronchial veins, whence it proceeds by the vena azygos into the vena cava. Another portion of the blood of the small bronchial veins flows within the substance of the lung into the pulmonary veins; hence, both in contraction of the auriculo-ventricular orifice impeding the outflow from the left auricle and in insufficience of the mitral valve, with systolic regurgitation from ventricle to auricle, and consequent impeded outflow to the contents of the left auricle and pulmonary vein, the natural and immediate effect will be, hyperæmia of the lungs (that is, capillary engorgement of the alveoli); yet, if the cardiac lesion be severe, bronchial catarrh not only is one of the most constant but one of the most physiological and inevitable symptoms which arise. We shall not be able to understand this condition, if we fail to keep in mind that a part of the blood of the bronchial mucous membrane flows into the left side of the heart, instead of the right.

Secondly. If the current of the blood in the great branches of the aorta encounter an obstacle, and more especially if the aortic stream itself be impeded below the point of origin of the bronchial arteries, the pressure of the blood is augmented in the other arteries which are not compressed or otherwise constricted, and hyperæmia ensues throughout the range of their capillary system. As an instance of this process (which *Virchow* aptly terms "collateral fluxion") we find sometimes that, in consequence of compression of the abdominal aorta by liquid in the peritoneum, or by accumulations of excrement or gas in the intestines, augmented pressure from within takes place in the bronchial and carotid arteries, resulting in "congestion of the brain or lungs." So also in the cold stage of intermitting fever the circulation encounters a material obstruction (throughout the entire periphery of the body), from the cutis anserina, and from the spasmodic contraction of the peripheral arteries. Perhaps this is the reason of the hyperæmia, and catarrh of the bronchial mucous membrane, which so often complicate intermitting fever, and of the cough which, in certain patients, is very distressing during the attack.

Thirdly. Irritants which act directly upon the mucous membrane, such as dust, vapors, too cold or too hot air, occasion in it hyperæmia and catarrh. Those who follow certain trades, as bakers, millers, and, above all, the stone-cutters, suffer constantly from this disorder.

Fourthly stands chilling of the external skin, and the action upon it of a sudden change of temperature. As we have already observed, we have no means of satisfactorily accounting for the phenomenon, almost daily observed, of a person suffering from bronchial catarrh after seating himself in a draught of air while perspiring freely. The process cannot be ascribed to collateral fluxion, since a mere change of temperature or exposure to a current of air, or to a degree of cold by no means severe, suffices to bring on the attack. In bleak, damp localities, particularly on the sea-side, such exciting causes are so numerous that bronchial catarrh is endemic.

Fifthly. It forms a symptom of typhoid fever, measles, and smallpox. Here it must be looked upon as the result of a morbid state of the blood, which has absorbed some deleterious material, with the nature of which we are unacquainted. However, although we cannot explain what we see upon physiological principles, there is something analogous to it in the symptoms of poisoning, which we can produce at will by the exhibition of an inorganic material (iodide of potassium) in large doses. As is well known, a very violent bronchial catarrh, often accompanied by an exanthema of the skin, not unfrequently arises during the employment of this agent, no other irritant having come into operation meantime either upon skin or mucous membrane.

Sixthly. Under the operation of unknown atmospheric or telluric influences, from time to time, epidemics arise, in which very extensive catarrh occurs, with unusually severe constitutional disturbance. It is doubtful if an infection like that of the acute exanthemata be the cause of this complaint, to which it bears a certain similarity. Such an epidemic, the "grippe," or influenza, appeared in the year 1732, and, travelling through Europe, from east to west, attacked at least one half of its population. The disease was a dangerous one, especially for children and for old persons, partly from the intensity of the fever, partly through extension of the local affection into the pulmonary alveoli, and partly from participation of the intestinal mucous membrane in the complaint, and from other complications. Since that time there have been repeated epidemics of influenza, particularly in 1800 and 1835. With this repeated reappearance of the malady, the vicious custom has gradually crept into use, both among physicians and people, of calling all the non-epidemic bronchial catarrhs influenza, when associated with violent general disturbance, and showing unusual obstinacy, or when the intestinal canal takes part in the disorder, using the term gastric influenza (*gastrische Grippe*). This bad custom has, in one respect, its advantage. No one suffering from a mere catarrhal fever is willing to consider himself seriously ill, or to keep his bed. For an attack of "influenza," however, he is content to lay up for a week or ten days.

Finally, we must observe that, in very many cases, the exciting causes of catarrh are unknown, unless we accept the explanation with which people usually content themselves, that "they must have taken cold somewhere."

ANATOMICAL APPEARANCES.—In the cadaver, acute catarrh of the trachea and bronchi leaves a redness, sometimes diffuse, sometimes mottled, the one being due to injection, the other to ecchymosis. The mucous membrane has a clouded look, is opaque, relaxed, and tears easily. This condition arises from infiltration occasioned by the augmented pressure of the blood within the capillaries. Owing to this œdema of the mucous membrane in which the submucous tissue participates, the calibre of the bronchi is reduced. The younger the subject, the smaller the calibre of the tubes, so much the more readily does tumefaction of the mucous membrane impede or prevent access of air into them, especially into those of the third and fourth magnitude. This is an important fact in symptomatology, particularly as regards the contrast between the symptoms of bronchitis in childhood and that in adults, and in the difference of the danger from the disease at the two periods.

At first the mucous membrane is dry, or covered with a scanty, tenacious, transparent secretion, containing but few young cells, and a very few mature detached cells of ciliated epithelium. Somewhat later an active development of cells usually takes place upon the surface, the product of which, when mingled with the secretion, now more copiously and freely poured out, imparts to it a turbid, yellowish appearance.

Upon opening the thorax, if the finer bronchi be obstructed or occluded by mucus, the lungs evince little or no disposition to collapse. Indeed, if the contraction or obstruction have attained a very considerable degree, the lungs bulge forcibly out of the opened thorax, so as almost to convey the impression that the chest was too small. And, in point of fact, the cavity of the thorax has not sufficed to accommodate the lungs without compression of the air contained in them. The inspiratory expansion of the chest has ceased with death, but the lungs have not been able to diminish in a corresponding manner, because the obstructed bronchi have not allowed the air to escape from the alveoli. This appearance has nothing in common with emphysema, with which it is frequently confounded.

Chronic bronchial catarrh usually presents to view a more intense brownish or dirty reddening of the mucous membrane. Its vessels are dilated, its tissue puffed and uneven, but, at the same time, more coherent and less easily torn. The mucous membrane itself is hypertrophied, and the fibrous longitudinal bands and muscular layer beneath the mucous membrane are still more so. As, simultaneously with this thickening, the elasticity of the mucous membrane and of the fibrous coat is lost, and as the swollen, sodden bronchial muscles are, in a great measure, deprived of their power of contraction, a diffuse dilatation of the bronchi, consequent upon relaxation of the bronchial wall, associates itself, in many cases, to chronic bronchial catarrh. This diffuse bronchial dilatation may become so considerable that, upon section, even small bronchi gape widely, exhibiting a larger calibre than the branches from which they spring.

Saccular bronchial dilatation, which is usually connected with important changes of structure in its surrounding pulmonary parenchyma, is again to be considered while treating of diseases of the lung, as is also emphysema, one of the common sequelæ of chronic bronchial catarrh.

In many cases of this disease there lies upon the mucous membrane a profuse layer of yellowish puriform secretion, containing great numbers of young granulated cells, with divided nuclei, while in others it is sparingly coated by a tenacious, glairy, semi-transparent substance. The latter form, in which the swelling of the mucous and submucous tissues is often very great, is called dry catarrh (*catarrh sec*). In the former the copious secretion often completely fills the smaller bronchi, while in the larger it is frequently mixed with air, and is frothy.

The diffuse and follicular catarrhal ulceration which we have described as occurring in the mucous membrane of the larynx seldom appears upon that of the bronchi. *Reinhard*, however, claims constantly to have noticed it in the finest bronchi, when surrounded by tubercular-degeneration of the lung substance, and believes that the disintegration of the tuberculous pulmonary tissue proceeds from this ulceration as soon as the wall of the bronchus has been destroyed, a view which we fully share with him as regards infiltrated tuberculosis (or cheesy pneumonia).

## I. ACUTE CATARRH OF THE TRACHEA AND LARGER BRONCHI.

SYMPTOMS AND COURSE.—Acute catarrh of the trachea and greater bronchi is often combined with catarrh of the larynx, nasal mucous membrane, that of the frontal sinus, and of the conjunctiva. More rarely it spreads into the smaller bronchi. The greater its extent, so much the more frequently does it begin with shivering, and, in sensitive persons, perhaps even with a chill. This chilliness, however, is rarely confined to a single rigor, and this is an important point of distinction between the onset of a catarrhal and of an inflammatory fever. It often happens that throughout the whole attack, with every slight alteration of temperature, or upon changing the linen, and especially if the patient be laid in a fresh bed, shivering-fits continue to recur. During the intervals the patient experiences a sensation of burning heat, without any indication from the thermometer of an actual increase of temperature. Added to this, there come distressing frontal headache, pulsation of the temporal arteries, soreness of the limbs, sensation of pain in the joints, increased upon pressure, constituting "catarrhal rheumatic fever."

Should the appetite fail, too, and the tongue become coated, we even hear "gastro-catarrhal-rheumatic fever" spoken of. It is very indicative of catarrhal fever that the intense feeling of weakness does not bear any proportion to the trifling elevation of temperature and frequence of the pulse, which seldom exceeds the number of eighty to a hundred beats per minute. In children, and irritable subjects, delirium sometimes is added to these symptoms, and in very rare instances, and in exceedingly sensitive children, convulsions may take place. The mother, and even the physician, may be temporarily alarmed by these manifestations, until a profuse flow from the nose, or repeated sneezing, allays the dread of a commencing disease of the brain. In such a case the doctor, who, the day before, with a long face, has been applying leeches to the head, may find his position embarrassing, now that the harmlessness of the case has become obvious, even to the bystanders.

Catarrh of the trachea and larger bronchi is not always attended by fever, often running its course from beginning to end without it. In most cases of severity it is accompanied by perverted sensations along the course of the air-passages and under the sternum. Hence the sensation of tickling after inhaling acrid vapor, and the feeling of soreness and burning in the chest. Not uncommonly, the integument of the sternum is unduly sensitive, and the cause of this phenomenon is supposed to be an excitement transmitted to the skin through central ganglia from the sensory nerves of the bronchial mucous membrane. The cough is not quite so distressing as that of catarrh of the laryngeal mucous membrane, which is so much more fully provided with sensory nerves. It never has a hoarse tone except when the larynx also participates in the disease. At first the sputa are scanty, or entirely wanting; afterward the expectoration becomes more copious; and as the secretion only proceeds from the larger bronchi, a few efforts suffice to cough it up. The patients say that the "cough is loose." In the beginning the sputa are transparent and viscid; at a later period they are turbid and yellowish.

There is, of course, no real dyspnœa in catarrh of the trachea and larger bronchi; or, at the utmost, there is but a slight oppression, and the patients say that "their chest is stuffed up." A swelling even of considerable magnitude, together with the most profuse secretion, is incapable of materially and injuriously diminishing the large calibre of these channels.

Percussion of the chest cannot show any change of sound during catarrh of the larger bronchi. The vibrations of the thorax and its capacity for air remain normal. Auscultation, too, in many cases, gives negative results—that is to say: we hear everywhere the whispering sound which the air flowing in creates at the points of division of the finer bronchi, and in the air-vesicles themselves, and which we call vesicular respiration. All thought of the graver catarrh of the minuter bronchi may be excluded, and catarrh of the larger tubes is to be diagnosticated, when we hear vesicular breathing alone in the chest of a person suffering from cough and expectoration. When the mucous membrane of the greater bronchi is considerably swollen at one circumscribed point, the air, passing through it as through a reed pipe, produces a buzzing, humming sound (*the sonorous rhonchus*), audible to the ear applied to the thorax, not only over the point of origin, but beyond, and often with perceptible vibration of the thoracic walls. If an accumulation of mucus afterward forms within the bronchi, the air sets the liquid in motion, or bursts through it, so that bubbles are formed and broken, causing rattling noises, which, as the bubbles are larger here than they can be in the finer tubes, we call large moist *râles*, to distinguish them from the *râles* produced in the lesser bronchi.

Catarrh of the trachea and greater bronchi, which we often hear called a "slight cough" by the laity, as a rule runs its course favorably, and with tolerable rapidity. The fever disappears, when there has been any; the cough, from time to time, particularly in the mornings, brings up sputa cocta, nowadays called muco-purulent homogeneous sputum, and finally subsides, the perverted sensation of the chest having previously vanished.

### II. ACUTE CATARRH OF THE SMALLER BRONCHI.

A. *As it occurs in the Adult.*—When extensive, the disease often exhibits the signs of sympathy and reaction of the general system against local disorder, already described as catarrhal fever.

The minuter bronchial tubes have no sensibility; hence, when the catarrh is confined to these alone, there is none of that feeling of itching, nor soreness, felt in catarrh of the larger bronchi. The occurrence of acute pain, at any time throughout the attack, shows the existence of a complication. After a while, however, pain is felt at the points of insertion of the muscles upon the chest and epigastric region. This proceeds from overstraining of the abdominal muscles, by whose spasmodic, jerking contractions the act of coughing is effected. This pain, which is observable elsewhere when muscles have been overstrained, is aggravated by any movement of the muscular fibres, but especially so upon coughing, and, during the fit, the sick man sits up instinctively, so as to relax the abdominal muscles. The cough is of far more violent character than that previously described, coming on in long paroxysms. It is not "loose," that is, the air, driven from the pulmonary vesicles by the spasmodic contractions of the chest, has difficulty in clearing the lesser bronchi of the secretion. Here, too, at first, the sputum is usually scanty; but it gradually changes, as described above, becoming more copious. As the sputum from the smaller tubes is unmixed with air, it is specifically heavier than water, and sinks in it; but, owing to its tenacity, it retains the shape of the tube from which it came, and by its adhesiveness it clings to the frothy, lighter secretion from the larger bronchi, which is mixed with air and floats. Thus the expectoration of acute bronchial catarrh of the smaller air-passages, when cast into water, forms a frothy layer upon the surface, with fine filaments hanging from it.

Extensive catarrh of the lesser bronchi is always accompanied by more or less dyspnœa. In adults, however, this rarely amounts to more than a somewhat laborious, or impeded respiration. Air enough can always reach the air-vesicles, and terror and sensation of suffocation are scarcely ever observed. Indeed, it is the mild and insignificant distress caused by this disease in adults, and the great danger and totally different symptoms to which it gives rise during childhood, which obliges us to describe the two forms separately. It is true that, even in adults, catarrh of the finer bronchi is sometimes accompanied by severe periodical dyspnœa; but this very periodicity indicates the existence of a nervous complication, which is causing spasm in the muscles of the smaller bronchi. The sound, upon percussion, is not altered in this form of bronchial catarrh, any more than in catarrh of the larger bronchi. Instead, however, of the sonorous rhonchi, sibilant rhonchi, whistling, wheezing sounds are heard in the narrower tubes from partial thickening of the mucous membrane. When secretion becomes more free, rattling sounds, or *râles*, are produced, and, as large bubbles cannot form in the small tubes, the *subcrepitant râles* are heard. In adults, catarrh of the finer bronchi also usually subsides in from eight to fourteen days. The fever ceases, the cough, the expectoration, and the slight dyspnœa disappear. In other cases chronic catarrh remains, but the disease rarely is dangerous.

If catarrh of the finer bronchi develop in an old man, or a younger person in very debilitated condition, and if it be accompanied by violent fever, the latter assumes an adynamic character, and symptoms attend it which indicate the assumption of a "nervous condition" (a very common term among the people). The sensorium becomes involved. Delirium or coma sets in; we notice the ominous dryness of the tongue, a symptom upon which we lay great stress, both for diagnosis and prognosis. The pulse is small, irregular, and very frequent; the skin, previously dry, flows with sweat; rattling noises arise in the chest (which may be coarse or fine, according as they occur in the greater or smaller bronchi), but which do not cease after coughing. At last the sound of still larger bubbles, tracheal *râles*, can be heard even at a distance. This gurgling (*kochen*) in the chest, which has also been called the "death-rattle," and during which the patient usually lies unconscious, indicates the approaching end. The older physicians applied the term "pneumonia notha" to attacks occurring in marasmic subjects, and which, originating usually in chronic disease, rapidly progressed to a fatal termination. Here the patient, in a few days, succumbs to a simple bronchial catarrh; and it is not the pernicious nature of the malady, but the peculiar condition of the patient, which leads to the danger. In old or enfeebled persons there is no disease, especially no inflammatory one, which may not threaten life. Fever, with its constant symptom of elevated temperature, the immediate cause of which is augmented combustion, or greatly accelerated consumption of tissue, rapidly consumes the scanty remnant of vital force which yet exists in these cases. There is nothing specific in any of these symptoms. They are repeated in exactly the same manner, wherever a febrile disorder is consuming the organism, and the physician does well in promising to the non-professional in these cases "that he can save the patient if 'nervous' (typhoid) symptoms do not arise." For hours and days before the end the function of the brain is disturbed, its nutrition being vitiated by overcharge of the blood with excrementitial products. The tongue becomes dry, the elevated temperature of the body causing an increased evaporation from the surface.

In almost all acute diseases, the pulse, toward the end, becomes small, irregular, and scarcely to be counted. In nearly all, too, the muscles of the skin are paralyzed, the skin becomes relaxed, and covered by profuse perspiration—the death-sweat. The bronchi, too, are provided with muscular fibre; the discharge of the secretion, which fills them, depends materially upon their contraction. If, too, these muscles are palsied with the others, the secretion accumulates. Œdema of the lung supervenes (see œdema of the lung), the palsied bronchi (not the palsied lung, as is often said) having lost all power to expel their contents, and thus finally the symptoms of suffocative effusion arise, which we have depicted above.

A chronic bronchial catarrh, which, as shown above, of itself relaxes the muscular element of the bronchi, must, of course, aggravate the peril in pneumonia notha, which is nothing more than a febrile bronchial catarrh in a marasmic subject.

B. *Acutè Catarrh of the Smaller Bronchi in Children.*—While bronchial catarrh of adults is a mild complaint, devoid of danger, and only perilous to old people on account of the accompanying fever, it is one of the most pernicious of diseases of childhood from causes purely physical. Let us first consider that intense form of catarrh to which a great number of children fall victims, especially during the period of dentition, and which may be called capillary bronchitis provided that this expression shall not be understood to mean a process differing essentially from catarrh, which is the basis of all the forms of disease hitherto described.

Sometimes the disease commences with the symptoms of a catarrh of the greater bronchi, apparently slight and free of danger; but, the further it descends into the smaller and finer canals, so much the more hinderance is there set up against the entrance of air into the air-vesicles. It is not limited in this case to that slight feeling of indisposition which adults experience in this disorder, but the feeling of oppression arises, and that nameless dread which always accompanies imperfect oxygenation and repressed liberation of carbonic acid. The same restlessness, the same violent efforts at inspiration, the same desperation which we have described in croup, are presented by a child, the tips of whose bronchi are contracted or closed by bronchial catarrh. As soon as we enter the chamber, and while still far from the bed, we hear the whistling, wheezing noise which the air produces as it is driven through the constricted canals. It accompanies both inspiration and expiration, and is easily distinguished from the whistling of croup; as we distinctly hear that, it does not proceed from one narrowed tube, but from many. If we husband the strength of the child and do not beset it with blood-letting and emetics, its strenuous exertion may often long remain adequate to the task of inhaling a proper supply of air to the air-vesicles. As the glottis is closed, the contents of the thorax compressed, the outflow of the jugulars restrained in this as in every other cough, the countenance becomes red, and even bluish, during the coughing-fits, which are exceedingly violent and distressing, seldom bringing up any secretion, and which, even then, is not ejected, but is swallowed by the child. Excepting during the paroxysms of cough, the color of the face remains normal. But, as the disease goes on (often through fault of the physician), if the child, either from exhaustion of its strength and inability to make further and adequate muscular effort, can no longer draw a sufficient supply of fresh air through the obstructed bronchi into the air-vesicles, or else because the obstruction has so increased that many of the tubes have become totally impervious to air, the picture changes. The previously full pulse now becomes small, the hot skin cool, the reddened countenance pale, the terror turns into stupor, and the well-known manifestations of carbonic-acid poisoning arise.

The first threatening of danger is not difficult to recognize, if we pay attention to the following points: As long as the epigastrium and the hypochondria remain prominent, air enough reaches the vesicles. If, however, instead of this, we see, at each inspiration, that the jugular and epigastric regions sink in, and that the lower ribs are drawn inward, we may feel sure that the air in the vesicles is being rarefied upon inspiration (as no new air can reach it), and that the respiratory function is being imperfectly performed. A symptom of imperfect inspiration of quite as much importance, and hitherto too little appreciated, or else falsely interpreted, is the prominence of the supra and infra clavicular regions, and the enfeeblement of the respiratory movement in this portion of the thorax. It often attracts the attention of the mother sooner than that of the physician, that a child, during its illness, has "got too high a breast." We must avoid confounding the permanent inspiratory expansion of the air-vesicles, to which this appearance is due, with vesicular emphysema. In this case the vesicles remain permanently in a state of expansion, such as they would attain normally at the height of inspiration. In emphysema they are abnormally distended, and it is curious that two such different conditions should hitherto have been so often confounded with one another. The manner in which the permanent inspiratory expansion of the vesicles takes place seems difficult of explanation upon a mere superficial glance at the mechanism of respiration. We are tempted to assume that the obstruction in the finer bronchi, which could be overcome by the action of the inspiratory muscles, should be still more readily conquered by forced expiration; since, as daily experience teaches, we are able to expel our breath with greater force than is required to draw it in, or, as the physiologists express it, the pressure of expiration is greater than the inspiratory pressure. But, if we keep in view the action of forced expiration, and the condition of the lung in intense and extensive bronchitis, the matter assumes a different aspect. In forced expiration we press the diaphragm upward by energetic contraction of the abdominal muscles, and thus exert a considerable pressure upon the lungs. This pressure acts as well upon the air-vesicles as upon the contracted bronchial tubes. The former cannot be cleared by the pressure, as this very pressure closes their outlets. Even when removed from the body, we are unable to diminish the volume of such a lung by squeezing it. I have been led to this simple explanation, by frequent observation that, in this class of patients every expiration is forcibly effected by means of the muscles of the abdomen, the sibilant rhonchus alone being audible at the time. If we lay the ear upon the thorax, we hear nothing but that disseminated sibilant rhonchus. Air enters in too small quantity, and, above all, too slowly to give rise to the whispering friction-sound which we call vesicular murmur. Where it exists it is overborne by the whistling sounds. Afterward we hear the wide-spread fine rattle, of minute bubbles (subcrepitant *râles*).

C. *Bronchial Catarrh of the New-born.*—New-born children very often contract catarrh of the respiratory passages, principally from inefficient protection against chilling during washing and bathing, etc. The symptoms of it have hardly any resemblance to those of the disease described above, although the malady is exactly the same. The complaint is almost always mistaken by unphysiological physicians, and regarded as an "organic affection of the heart, in consequence of which the child is fortunate in dying so soon." In such cases children have sneezed a few times, have perhaps coughed a little, otherwise appear well, and often inclined to sleep. The parents rejoice over their quiet, contented babe; they do not notice that it only breathes superficially. The physician is not called in, or, if he comes, he finds no occasion to take account of the breathing; but a great change comes over the child, usually with suddenness. The face becomes pale, or, together with the whole body, changes to an ashy hue; the nose grows peaked, the eyes dull, the arms and legs hang down inertly. The temperature of the surface is lowered, and unequal; an acute cyanosis is diagnosticated, an unhappy conception, wherein extreme vascular engorgement is confounded with the condition which imparts the bluish hue to the lips of the dying, and which is really owing to the beginning of that contraction of the arteries and discharge of their contents into the capillaries and veins which we always find *post mortem.*

The explanation of these symptoms is easy. As long as only "snuffles" and catarrh of the larger bronchi exist, the illness is indeed trifling. By-and-by the catarrh spreads to the finer bronchial tubes. These, very small by nature, are easily closed. The as yet undeveloped child is unable to make powerful efforts to overcome the obstruction. There is no rhonchus sibilans, which otherwise would occur, to afford a diagnostic mark. Nor does the child cough as often as an older one would do, for a portion only of a cough is involuntary; the other part is voluntarily made in order to remove impediments to respiration. Experience has not yet taught the child this art, and so the symptoms of carbonic-acid poisoning arise both suddenly and unexpectedly as soon as the minuter bronchial tubes become involved in the catarrh. In the bodies of such children, we often find the air-vesicles collapsed, to which the obstructed bronchi led. (Atelektasis, see disease of the lung, Chap. II.) The described symptoms, however, are just as capable of occurring without collapse of the lung, and the latter, as well as carbonic-acid poisoning, is a consequence of bronchial catarrh.

If we can succeed in making the baby cry or vomit, and thus bringing about energetic respiratory movements, fresh air once more enters the lung. The carbonic-acid poisoning vanishes, the symptoms of palsy cease, and as the heart now contracts again with vigor, blood anew streams from the veins into the arteries, and into the empty heart. Circulation is reëstablished, and with it normal color and warmth return to the skin.

The attacks are very apt to recur. It may not be possible a second time to render the air-passages pervious, and the children may perish in a subsequent seizure. If the autopsy be conducted without care, or science, the cause of death often remains concealed, unless accompanied by extensive atelektasis; since an accurate examination of the bronchi is not attempted, the more ordinary symptoms of bronchitis not having been observed during life.

### III. Chronic Bronchial Catarrh.

The symptoms which we have ascribed as belonging to acute bronchial catarrh are only slightly modified in chronic catarrhal bronchitis, an extensively prevalent disease. The seat of the affection is not usually confined to solitary portions of the bronchial mucous membrane, as in the acute form, but the anatomical changes given above reach from the trachea, in greater or less degree of development, into the ramifications.

The disease almost always develops from a frequently relapsing and protracted catarrh, which has habitually recurred every spring and fall. At first, during the summer, the patients remain exempt, until, finally, the symptoms become perennial, although somewhat moderated in intensity. The most violent distress arises from that form of the disease in which a gray tenacious secretion lies upon the mucous membrane, which is particularly swollen in such cases, while chronic bronchial catarrh, with more copious and more liquid yellow secretion, occasions comparatively slight trouble. In the former variety (catarrh sec of *Laennec*), it is the protracted and tormenting coughing-spells (due to the toughness of the secretion, and its situation in the smaller bronchial tubes) which are the chief source of suffering to the patient. During these paroxysms of coughing, the interruption to the current of the jugulars causes them to swell greatly, the countenance becomes dark-red and bluish, the eyes weep, the nose drips, "the head seems as if about to split," and is spasmodically grasped by the patient with both hands. Not unfrequently the veins, distended by constant coughing-fits, remain varicose, even where there is no emphysema, and thick blue vessels show themselves on the cheek and alæ nasi. An attack of coughing, of great violence, frequently terminates in retching or vomiting, the contents of the stomach being pressed out by the contracted abdominal muscles.

Permanent dyspnœa is much more common in this disease than in acute catarrh, since in this the mucous membrane is more thickened and swollen, and thus offers greater impediment to the entrance of air. When, as often happens, an acute attack of bronchial irritation sets in upon a chronic catarrh, the dyspnœa becomes much aggravated, the affection receives the name of humid asthma. After a short walk in a cold, dry atmosphere, which seems particularly hurtful to such invalids, they often remain for weeks in the most miserable condition, obliged constantly to sit upright, and to pass even the entire night in an arm-chair, in order to aid the expansion of the chest as much as possible.

In consequence of the dyspnœa and of the continuous and immoderate exertion of the inspiratory muscles induced by it, the latter become hypertrophied. This hypertrophy is most marked in the sterno-cleido-mastoid muscles and in the scaleni, which stand out upon the neck like strong cords. Like other hypertrophied muscles, those of the respiratory apparatus are constantly in a condition of moderate contraction; and (just as locksmiths or blacksmiths and the like habitually carry their arms slightly flexed, instead of letting them hang loosely), so in chronic bronchial catarrh, the chest is as it were drawn up toward the head by these muscles. The neck seems shorter and thicker, the chest more convex; but we are not warranted in diagnosticating the complication of emphysema of the lungs from these symptoms alone, although the complication is common enough.

Sometimes, during long-protracted and severe exacerbations of bronchial catarrh, the jugular veins become distended, cyanosis appears, and not unfrequently there is general dropsy. As both cyanosis and dropsy vanish as the attack subsides, and the jugulars become unloaded, there can be no doubt but that the symptoms were really due to the catarrh itself, and not to any complication. Their occurrence is by no means difficult to account for, if we can only prove to ourselves that patients, the calibre of whose bronchial tubes is considerably reduced, always contract their abdominal muscles during the act of expiration. Thus, as the exit of the air from the vesicles through the narrow tubes is slow, a considerable pressure is exerted upon the blood within the thorax, and the flow of blood thither from the rest of the body is retarded. Hereafter, when treating of emphysema, we shall show that bronchial catarrh is one of the main causes of cyanosis and dropsy in that disease.

Alteration in the percussion-sound, if it exist in chronic bronchial catarrh, is never on account of the disease itself, but is due to emphysema, one of the most frequent of its sequelæ. Upon auscultation, we often hear the sibilant rhonchus, more rarely the sonorous rhonchus, in other cases small, moist *râles* (subcrepitant *râles*). At the same time there may be normal vesicular respiration, or, as sometimes happens, when many of the bronchioles are occluded, the respiration is feeble; and again, where the swelling of the mucous membrane has diminished, but has not closed the bronchioles, thus increasing the difference between their calibre and the capacity of the air-vesicles, the respiratory murmur is sharper.

Few patients ever recover from this malady, yet to very few does it ever endanger life. The old man's cough has become proverbial, and, indeed, these patients may attain a very great age ere they succumb to pneumonia notha, or other intercurrent malady. In other cases they die of the secondary disorders of the pulmonary substance which result from chronic catarrh. (See article on emphysema and interstitial pneumonia.) A very different character from that of the "catarrh sec," with its more or less tough, scanty mucous secretion, is presented by the variety of bronchial catarrh attended by copious secretion, and often called bronchial blenorrhœa, or bronchorrhœa. In this form the sputum is in coherent masses, which, more or less mixed with air, do not sink in water. Sometimes a pound or more of this yellowish secretion, full of young cells, is coughed up in the course of a day. In winter it is profuse; in summer it usually diminishes in quantity.

The secretion being less adhesive, and hence more easy of expectoration, the cough is not so persistent and distressing as in the "dry catarrh." The dyspnœa, too, generally is slighter, the more common seat of bronchorrhœa being in the larger tubes, and its tendency being rather to produce diffuse dilatation of the finer bronchi. It is only when an acute attack supervenes upon the chronic one that there is much dyspnœa, which then depends upon the amount of swelling of the mucous membranes.

During these exacerbations the cell-production goes on with less activity, so that the secretion of the mucous surface is reduced in quantity. This causes the patient to imagine that the expectoration has become "tight, and must be loosened," a view in which, here and there, the doctors participate, who, upon increase of the dyspnœa, with arrest of secretion, forthwith diving into their arsenal of expectorants, compose a recipe of the most heterogeneous substances.

Upon auscultation, in this form of chronic bronchitis, we sometimes hear the coarse rhonchi, sometimes coarser, or finer *râles*.

This disease, too, upon the whole, is wonderfully well borne. The patients not unfrequently attain an advanced age ere pituitary catarrh, phthisis pituitosus, develops from blennorrhœa of the bronchi. While the dry catarrh is more prone to the production of emphysema of the lungs, the tendency of bronchorrhœa is to cause bronchiectasis. The patients more frequently die of acute intercurrent disorders than of exhaustion through the persistence and abundance of the discharge.

The general characteristics of chronic bronchorrhœa are not, in all cases, nor even in the majority of cases, so much modified by diffuse dilatation of the bronchi as to enable us to recognize this complication with certainty. Sometimes, however, the peculiar nature of the sputa warrants our forming a diagnosis at least of its probable existence. Experience has taught that, as long as the bronchi retain their normal calibre, the secretion of their mucous membrane seldom undergoes putrefactive decomposition, while in the diffuse and sacculated bronchiectasis it very often becomes putrid. The fact that the contents of a sacculated bronchus frequently putrefy, far more frequently, indeed, than the contents of a tuberculous cavity, is a matter for our future consideration.

That the secretion formed in tubes which have become diffusely enlarged should also show an increased tendency to putrescence would seem to indicate, with some plausibility, that besides the ciliary motion and the cough (which appears to have little effect in clearing the minuter bronchi), contraction of the bronchial muscles also plays a part in expectoration, so that palsy of these muscles, which, undoubtedly, is one of the main causes of dilatation in the tubes, also favors stagnation, and consequent putrescence of the secretion which they contain. If, then, the copious puriform sputa hitherto ejected become unusually liquid; if both sputa and the breath of the patient begin to emit a penetrating stench; if the more solid components of the sputa sink to the bottom of the cup, and there form a greenish-yellow sediment, being no longer held in suspension by the decomposed mucous, which has lost its tenacity; if, finally, we find in the sputum a few inspissated whitish, cheesy plugs, of particularly evil odor, we may infer the existence of a bronchiectasis with great confidence. The microscopic examination of this sputum, which, curiously enough, is often less offensive in the vessel than at the moment of its ejection, shows it to consist in part of young, well-preserved cells, in part of cells in a state of fatty metamorphosis, with some masses of detritus, an appearance which is found elsewhere, where pus cells have long lain stagnant. Not uncommonly, too, we find very delicately-shaped objects in the cheesy masses, tufts of fine needles, which prove, upon employment of ether, etc., to be fat-crystals (margarine and stearine), and which are observed in the sputa of gangrene of the lungs, as well as in that of bronchial dilatation. Absolutely certain diagnosis, however, of one form or other of dilatation of the bronchi (of whose differential diagnosis we shall treat hereafter), cannot by any means be established by the character of the sputa. In some instances, as has been proved by *Traube*, the bronchial secretion takes on a similar character, without the existence of any bronchiectasis. This putrefactive decomposition of the bronchial contents often has a very prejudicial effect upon the wall of the tube, and the adjacent parenchyma of the lung. In treating of gangrene of the lungs, we shall find that this putrescence of the contents of the bronchial tubes is one of its most common exciting causes. In other instances, which, indeed, are even more common, it results in the development about the bronchus, or even throughout an entire pulmonary lobe, of an extensive pneumonia, with a soft, easily-liquefying exudation.

DIAGNOSIS.—The distinction between simple acute bronchial catarrh and catarrh of the larynx is easy. Hoarseness of the voice and of the cough always indicates the latter, and that swelling of the mucous membrane has extended to the vocal chords.

The points of distinction between acute bronchial catarrh and acute disease of the pulmonary parenchyma can be discussed to greater advantage after we have studied the symptoms of inflammation of the substance of the lung. For the present, we shall merely indicate a few important diagnostic points, which we already have had occasion to speak of while detailing the course and progress of the disease.

1. Simple acute bronchial catarrh is never accompanied by darting pain in the side. The only painful sensation proper to it is a feeling of soreness and burning in the chest, and sensibility at the points of insertion of the abdominal muscles upon the thorax. When other pains arise, complications always exist.

2. Acute bronchial catarrh, of itself, never changes the sound of percussion, so that the presence of the physical signs which indicate

condensation of the parenchyma of the lung exclude simple catarrh from the diagnosis.

3. It is true that acute bronchial catarrh may begin with a violent rigor, but, as the disease progresses, the tendency shows itself to repeated attacks of chilliness. Hence, when we find an asthenic fever without pain in the side, without bloody sputa, and apparently with none but catarrhal symptoms, yet, if the attack have commenced with but a single rigor, we should not be too hasty in diagnosticating a pneumonia notha, or, as we now say "nervous influenza" (typhoid influenza), lest autopsy should bring to light a pneumonia which would have been recognized, had the single rigor been properly considered and the patient been more accurately auscultated.

In their appropriate chapters we shall explain the mode of distinguishing chronic bronchial catarrh, with scanty secretion and great dyspnœa, from nervous asthma, and shall give the diagnostic points between pituitary phthisis and tuberculous phthisis.

PROGNOSIS.—The danger of bronchitis depends almost entirely upon the age of the patient. In early life, the younger the child, the smaller the bronchi, so much the more perilous is the disease. In adults, it scarcely ever threatens life, excepting among old persons, where it again becomes a dangerous disorder, especially if accompanied by fever.

The gravest symptoms are those indicative of impeded oxygenation in the air-vesicles. The most violent cough, the most intense restlessness, an immense purulent expectoration, and all other symptoms of the acute and chronic form, are of far less significance than the first token of poisoning by carbonic acid. Never forget that life is not really threatened until such symptoms arise. This reflection will form the best safeguard against over-active treatment of children with capillary bronchitis. As long as the pulse remains full and the countenance ruddy, there is no immediate danger.

TREATMENT—*Prophylaxis.*—Upon this head we refer to what has been said as to the prophylaxis against laryngeal catarrh. Cautious habituation to change of temperature, cold washing, cold baths, are as commendable in the one case as in the other.

*Indication as to Cause.*—This demands consideration both of the predisposing and of the exciting cause. As some of the causes of catarrh are unknown, and as others cannot be allayed, the *indicatio causalis*, in many cases, cannot be met; while, in other instances, careful regard to known exciting agents is rewarded by the best results.

This applies, above all, to the general tendency to catarrh, and especially to bronchial catarrh, engendered by scrofula, and rachitis. There are many pigeon-breasted, big-headed children, with open fontanelle, enlarged epiphyses, retarded dentition, and flabby skin, which flaps like a pair of loose breeches against their bones, who for months suffer from bronchial catarrh, and who are often supposed to be tuberculous. Expectorants and derivatives are useless here; but if we place such children upon a well-selected diet, giving them milk and underdone meat, if we prescribe cod-liver oil and salt baths, the results are often brilliant. The children recover, and nothing but the pigeon-breast remains to recall to mind the serious illness of childhood. Again, we have found the malady to be a very common one in advanced life, but particularly so among a class of people of about fifty years of age, "high livers," who drink freely of wine, sit all day, assimilating much material, and consuming but little; with hæmorrhoids and a voluminous paunch, who evince a great tendency to chronic affections of the abdomen, as well as to chronic bronchial catarrh. It were folly to confine such a person to his room, and set him to taking Seltzer-water and milk, sulphuret of antimony or senega. Let him rather institute a proper proportion between assimilation and consumption of nutriment, cause him to take exercise, forbid spirituous liquors, and set him upon a sparing vegetable diet. Finally, let a patient of this class betake himself to Marienbad, Karlsbad, or Kissengen. In such cases, but only in cases like them, the alkaline chalybeates have a beneficial effect, not upon the cough alone, and other symptoms of catarrh of the bronchi, but upon the corpulence and the hæmorrhoids.

Among the exciting causes, mechanical obstacles at the mitral valve, which impede the venous circulation of the bronchi, sometimes admit of palliation. When the catarrh depends upon insufficience of the mitral, the action of digitalis is uncertain, but its effect is very evident where the hyperæmia is due to its contraction. In the latter case, by retarding the action of the heart, time is afforded to the auricle to discharge its entire contents into the ventricle, the engorgement of the pulmonary vein subsides, and with it the bronchial catarrh to which it has given rise.

Bronchial catarrh, caused by the collateral fluxion to the lung in malarious fever, requires quinine. The collateral fluxion into the bronchial arteries, produced by the pressure of a dropsical effusion upon the abdominal aorta, may demand tapping; the more so, as the diaphragm, which in these cases is pushed upward into the chest, compresses a portion of the lung. After having once witnessed the striking amelioration effected by tapping, perhaps even the complete subsidence of a bronchial catarrh, which a few days before was the patient's most grievous affliction, we shall never permit any case of catarrh whatever to be aggravated by collateral fluxion, the result of pressure upon the abdominal aorta by accumulated fecal matter or by gas, the removal of which is still more easy. A teaspoonful of the pulvis liquiritiæ compositus, taken morning and evening so as to produce a sufficient daily alvine evacuation, is a remedy much praised by the patient, and readily taken.

If the cause be a direct irritant to which we know the mucous membranes are daily exposed in certain trades or avocations, the causal indication cannot be met, as these patients are not usually in position to give up their occupations, and to avoid these noxious agents. As we have explained above, chronic bronchial catarrh is undoubtedly exacerbated if the patients subject themselves to the action of very cold, dry air. We should bear this knowledge conscientiously in mind, and make the patient keep his room for weeks or months during a cold winter, and establish a uniform temperature in his chamber. Experience demands our recourse to this procedure all the more, as patients with chronic catarrh of the bronchi, after suffering an intercurrent acute attack, often indulge in the idle hope that the disease just passed away has had some critical influence upon the chronic evil, and that they now cough much less, and are much less oppressed about the chest, than before.

Their error becomes evident as soon as they expose themselves anew to the air. Chronic bronchial catarrhs, which have arisen under the influence of a severe climate, require, where circumstances permit, a change of abode. Let the patient avoid the winter—that is, send him, during the cold season, into some milder climate. During spring and autumn advise residence in some peculiarly-sheltered place, Baden-Baden, Wiesbaden, Soden, etc., or in the highly-oxygenated atmosphere of the pine-woods, in which convenient accommodations have long been established for the "pine-needle-bath institutions." As a rule, high dry places are more suitable for the catarrhus pituitosus, while we must send patients who suffer from the dry catarrh to the wooded coasts, or to promenade upon the salt-works. In epidemic catarrh, the cause cannot be obviated.

INDICATION IN TREATING THE DISEASE.—Even the mighty bleeders of the school of *Bouillaud*, who make little of a pound or two of blood, claim nothing for blood-letting in catarrh of the respiratory organs. Let us bear this fact in mind, lest we be induced to bleed at the sight of severe infantile dyspnœa, from hyperæmia, and swelling of the bronchial mucous membrane; and let us also remember that the danger from the so-called capillary bronchitis of childhood arises merely from the situation of the disease. In the vast majority of these cases, and it is in these alone, of the many forms of bronchial catarrh, that we might be misled into bleeding, instead of averting the danger of carbonic-acid poisoning by so doing, we should enhance it. The swelling of the mucous membrane will not subside, and although hitherto, by dint of strenuous efforts, the child may have been able to draw air enough through its narrowed bronchi to support life, yet after the bleeding his strength may be inadequate to the exertion. He who has once seen the altered aspect of a child a few hours after such a bleeding, and, on the other hand, has had opportunity of observing how long the undepleted powers of nature are able to sustain a respiration, which, if laborious, is still sufficient, will readily abstain in these cases from venesection.

The "antiphlogistic" neutral salts of potash and soda are as little employed in catarrhal inflammation as depletion by the lancet. Calomel, also reckoned an antiphlogistic, is extensively used, both in the bronchial catarrh of teething children and the catarrh of the intestine which develops about this period. Incomprehensible as the beneficial effect of this drug upon either of these disorders may be, yet experience has affirmed it so fully that we cannot have any hesitation in making use of the remedy. We give small doses of from the sixth to the quarter of a grain, three or four times a day. Certain salts, to which there has been ascribed less of an antiphlogistic than of anti-catarrhal virtue, have come into very extensive use in bronchial catarrh, either because they excite the action of the skin, or because they are supposed to directly modify the nutritive condition of the mucous membrane of the bronchi. Among these are certain antimonial preparations, golden sulphuret of antimony, Kermes mineral, tartar emetic, and, above all, muriate of ammonia. The mistura solvens, which consists of muriate of ammonia and liquorice āā (ʒ j), with one grain of tartar emetic, or one or two drachms of antimonial wine, dissolved in six ounces of water, forms almost a third of all the prescriptions which come into the apothecary's shop. When I consider that physicians, and even very clever ones, devoutly order a tablespoonful every two hours of this nauseous dose, and even take it themselves upon occasion, I hesitate to declare that it can hardly have any other effect than to irritate the gastric mucous membrane and to embarrass the digestion. Perhaps from the sal-ammoniæ and the antimonials some slight palliative action might be expected in cases where the mucus continues to retain an abnormal viscidity.

The treatment by diaphoresis is highly to be recommended where the catarrh is recent, and particularly when cold is the assignable cause. Whether determination to the skin act as a derivative to the vessels of the mucous membrane, or whether the beneficial action arise from other influences, their happy effect upon recent catarrh is established by brilliant experience. The irritability of the mucous membranes can be diminished even in a few hours, and in fortunate cases, by profuse sweating, we may even succeed in cutting short the catarrh. It seems a matter of indifference how we produce the diaphoresis. Copious potations and warm bed-covering seem to be the most sure means. It is doubtful, to say the least, if the flores sambuci, spiritus miridereri, antimonial wine, and other so-called diaphoretics actually do have a diaphoretic effect; nay, it seems to be of no consequence as to the result, whether we cover up the patient in a good bed and in warm blankets, or whether we wrap him closely in cold wet clothes and then cover him up, as these cold applications, from the retained heat of the body itself, are very soon converted into warm ones. Perhaps a stronger fluxion to the skin is produced by the latter process than by the former. Upon similar principles, in chronic catarrh, even where there is no scrofulous nor rachitic taint, determination to the skin by the use of salt baths is indicated, particularly such baths as contain an extra amount of brine, like those of Kreutznach. This treatment is particularly adapted to the cases of all patients in whom a protracted acute bronchitis is threatening to become chronic, but in whom it has not as yet assumed an inveterate character. But, even in inveterate and grave cases of chronic bronchial catarrh, I have produced most striking results, at my clinique, by means of energetic diaphoresis. I have kept patients for half an hour at a time in a bath of a temperature of at least 100° Fahrenheit, and then enveloped them in hot blankets, in which they remained from one to two hours longer. At first, as long as the dyspnœa was very severe, the patient suffered greatly, not only while in the bath, but during the sweating. Soon, however, generally toward the end of the first week, as the perspiration began to flow more readily and freely, they became satisfied at their improved condition, and were willing to continue the treatment. After eight or ten baths the dyspnœa had abated in the most striking manner, and the cyanosis had disappeared.

Rash as it may seem to persons unfamiliar with this mode of treatment, to plunge a patient, panting with dyspnœa and blue with cyanosis, into a hot bath, yet such of my pupils as have witnessed the results of the procedures described above will be more successful than such as fear to resort to forced diaphoresis in treating this distressing malady, which often mocks all remedial measures.

Allied to treatment by general diaphoresis, there is a class of remedies by means of which a sort of local diaphoresis, or, at all events, a local derivation, is set up upon the integument of the chest, and in whose favor experience speaks strongly. In cases of protracted catarrh of the bronchi, make the patient wear flannel; put a pitch-plaster upon his chest. We must not, however, be too hasty in the employment of blisters and of sinapisms, the use of which is not indicated by the catarrh itself, but only by certain symptoms. While fever exists, they are best omitted.

The introduction of the inhaling apparatus has resulted in a considerable advance in our mode of treating bronchial catarrh. We refer the reader to what has already been said upon this subject in treating of disease of the larynx.

SYMPTOMATIC INDICATIONS.—We shall now endeavor to lay down rules for the exhibition of the so-called expectorants, as we believe that the acceptation of this word is somewhat indefinite.

As we have seen, the abatement of the hyperæmia and a favorable termination of the disease are announced by an increase in the amount of liquid secretion and by a more copious production of young cells. These symptoms, however, are the result, not the cause of the improvement; hence the formation of *sputa cocta* will be best promoted by any means which tend to bring about resolution of the catarrh. Then, too, when there is an accumulation of sputa in the bronchi, their expulsion may be hindered by so many different causes, that the various remedies, which may be useful in aiding the expulsion, cannot all be placed in the same category. The following are the most important symptoms which must be treated in accordance with the pathologico-anatomical phase of the disease:

1. We have to do with catarrhs in which there has long existed an excessive irritability of the mucous membrane, so that the patients are tormented by incessant and most distressing cough. Although these coughing-fits are only a result of catarrh, yet, from the friction of the air upon the irritated mucous membrane, which they occasion, they are also one of the causes of its aggravation and persistence. If, then, we combat these paroxysms, we not only moderate the individual evil, but tend to cut short the general course of the disease. The sweet, mucilaginous decoctions and syrups are here both inefficient and injurious to the digestion, as are also the cough-bonbons and caramels, in spite of the illustrious names on the labels which testify to their efficacy. On the other hand, the employment of the alkaline muriatic mineral waters of Selters, Ems, Obersaltzbrünnen, is here as urgently to be recommended as the folly of resorting to them in every bronchial catarrh, as in a bronchial blennorrhœa, is to be decried. Of these waters, of whose beneficial action upon the irritable mucous membrane we have no physiological explanation, let five or six glasses be drunk fasting in the morning, while promenading; or, in acute catarrh, let them be taken in the course of the day instead of the customary ptisane. Be bold, too, in administration of narcotics under these circumstances. If rest be disturbed at night, give ten grains of Dover's powders in the evening; or, when incessant coughing torments the patient, administer, through the day, a mixture containing opium or morphine. The cough will then be less frequent, and the secretion from the bronchi, having more time for accumulation, will, when expectorated, appear in greater quantity, so that the patients are apt to praise the powder, which "has loosened their

cough so well," as the best they have hitherto taken. The application of strong, cutaneous irritants, sinapisms, and blisters to the chest, is especially adapted to the form of disease in question.

2. Sometimes a periodically recurrent dyspnœa, accompanied by very extensive sibilant rhonchus, forces us to the supposition that the muscles of the finer bronchi are in a state of spasmodic contraction, and that a part of the dyspnœa is to be attributed to this circumstance. For these cases, which often are accompanied by the excessive irritability of the mucous membrane just spoken of, narcotics are also serviceable in order to relax the tension of the bronchial muscles. Besides these, however, we can here have recourse to the nauseants with decidedly favorable results. They seem to relax the bronchial muscles just as they produce relaxation of the muscles at large. We here prescribe weak infusions of ipecacuanha, small doses of tartar emetic, and, perhaps, the mistura solvens owes to its nauseating action the relief which it now and then affords to the dyspnœa.

3. There are other instances in which an opposite condition prevails, the bronchial walls being relaxed, the bronchi dilated, and their muscular coat half paralyzed. It is in such cases, above all, that the secretion is apt to be profuse, and, owing to the imperfect coöperation of the bronchial muscles, difficult to get rid of by coughing. (As already stated, large and soft moist *râles* indicate blennorrhœa of the bronchi.) The appropriate remedies, in such cases, are the stimulant expectorants, an important class among the so-called expectorants: senega, squills, pimpinella, carbonate of ammonia, camphor, benzoin, liquor ammoniæ anisatus. A very favorite prescription is an infusion of senegæ ʒ ij—℥ ss to water ℥ vj, with liq. ammon. anisat. ʒ ss. The elixir pectoralis, consisting of sacc. liquiritiæ ℥ ij, aqua fœniculi ℥ vj, liq. ammon. anisat. ℥ ij, to be taken by the teaspoonful. Finally, the use of ptisanes, containing more or less of aromatic substance, is suitable in these cases, although their virtue lies mainly in the warmth they give out when swallowed while very hot. Preposterous as it may be to prescribe pectoral teas to all classes of patients, those who are suffering from bronchial blennorrhœa with relaxed bronchial muscles almost always expectorate with greater ease after having drunk a few cups of hot ptisane. Besides the officinal expectorants, a mixture of mucilaginous, sweet, demulcent (soothing?) articles are ordinarily prescribed as a pectoral tea: rad. althæa, flores malvæ, flores et herba verbasci, radix liquiritiæ, with the seed of plants which contain an ethereal oil, such as semina anisi vulgaris, sem. anis. stellati, semina fœniculi, semina phillandrii aquatici. The latter article, which also contains resinous matter, and therefore belongs among the remedies to be spoken of in the next class, is in especial repute in bronchorrhœa, in which it is said both to facilitate the expectoration and to

restrain the secretion. The stimulants excite the bronchial muscles to more energetic contraction, just as they cause the pulse to rise and the heart to beat more vigorously. But it may happen that the bronchial muscles become so debilitated as no longer to afford any assistance in expectoration, and the cough alone is inadequate for the expulsion of the sputa. This state has been called "incipient palsy of the lung," but the lung takes no active part either in inspiration or expiration. It may be detected when, immediately after the act of coughing, the *râles*, instead of subsiding for a time, persist with scarcely any diminution. In such an emergency, should the expectorants fail, an emetic is imperatively indicated. Let no time be lost, lest the access of air to the alveoli, being cut off by the accumulating secretion, and the bronchial palsy already commencing, be aggravated by carbonic-acid poisoning. An emetic is the surest expectorant. If, during retching, the abdominal muscles be energetically shortened, and the thorax powerfully contracted, the air, which, as we have already explained, is expelled in violent puffs, drives the secretion out of the bronchi, or at least out of the larger of them. Unfortunately emetics fail us altogether where we have to overcome obstruction in the finest bronchial tubes, and this it is which renders the latter stage of capillary bronchitis so dangerous. Even firmly-adhering croup membranes are often enough loosened and expelled by the act of vomiting; but an inspiration of twice the force of that which accompanies this act is incapable of driving a current of air into the smallest bronchi and of forcing out the mucus contained in them. On the other hand, compression of the air-vesicles thus produced is accompanied by an equally firm compression of the capillary bronchi, by which they are still more firmly closed.

4. In the treatment of symptoms it may become our task to moderate the redundant bronchial secretion, which threatens to exhaust the patient. A great part of the means recommended for this purpose, the lime-water, acetate of lead, tannin, ratanhia, foliæ uvæ ursi, are either inert or of doubtful efficacy. The resins and balsams, recommended upon the same ground, are more serviceable in diminishing mucous secretion, of which their efficience in treatment of gonorrhœa furnishes striking proof. Peruvian balsam, balsam copaibæ, myrrh, gum ammoniac, belong to this class. A very favorite prescription is the Griffiths mixture:

℞ Myrrha pulv. ʒj.
Potas. carb. et tart. gr. xxv.
Aqua menth. crisp. ℥viij.
Ferri sulph. crystal. ℈j.
Sacc. alb. ℥ss.
M.
S., a tablespoonful four times a day.

It is naturally to be desired that these remedies shall come in as direct contact with the bronchial mucous membrane, as they do with that of the bladder or urethra, upon which they act immediately, when excreted into the urine. Here saccharum myrrhæ, taken dry, is recommended. We may calculate upon a small portion, at least, of the drug administered in this way, passing the glottis and reaching the trachea and bronchi. The application of remedies in the form of gas is far more serviceable. Thus we may boil tar either alone, or mixed with water until the atmosphere is entirely impregnated with its fumes; or we may put half a drachm of turpentine in a bottle of hot water, and cause the patient to inhale, for a quarter of an hour, four times a day, through a mouth-piece, attached to the neck of the bottle. Of course we can only expect success to follow this treatment when we apply it to the cases to which it is adapted; that is, where the mucous membrane is the seat of excessive purulent secretion. In all other forms of bronchial catarrh it does harm. The bitters and tonics which are also given, especially in bronchorrhœa, and among which are the polygala amara, the lichen islandicus, the folia cardui benedicti, may have an influence upon the mucous membrane of the stomach, or may improve the appetite, regulate the digestion, and act beneficially upon the nutrition of the body, which is then better able to bear the disease. Upon the disease itself they scarcely have any material influence.

5. In the treatment of bronchial catarrh in young children, who do not know how to cough, and who are unequal to the emergency when an obstacle in their air-passages requires a corresponding respiratory effort, the symptomatic indication requires, first of all, the removal of the accumulated secretion by means of an emetic, and secondly measures which shall force the child to more vigorous respiratory efforts. Do not let him sleep too much, and too profoundly. Put him in a bath, and spirt cold water upon his chest while in the bath. Make him scream by brushing the soles of the feet, if symptoms of obstruction of the bronchi and embarrassed oxygenation in the vesicles set in.

## CHAPTER II.

### CROUPOUS INFLAMMATION OF THE TRACHEAL AND BRONCHIAL MUCOUS MEMBRANE.

Etiology.—Not unfrequently, croupous laryngitis spreads into the trachea and bronchi; and in like manner we shall find that croupous inflammation of the pulmonary vesicles almost always is continued into terminal branches of the bronchial tubes. Besides this, however, there occurs, although rarely, a croupous bronchitis, which appears primarily in the bronchi of the third and fourth order; and, as this constitutes a distinct disease, it must be spoken of separately.

This primary croupous bronchitis attacks by preference persons during the age of adolescence, but we have not as yet any accurate knowledge as to either its predisposing or exciting causes.

ANATOMICAL APPEARANCES.—The tree-like ramified tubes, already described, are formed by the extension of the croupous process from the larynx into the trachea and commencements of the bronchi. The croupous plugs which fill up the bronchioles in pneumonia we shall find to be a constant feature in the sputa of pneumonic patients.

In primary independent croupous bronchitis the same condition of the bronchial mucous membrane is found, and with the same coagulated exudation upon it which we have described as existing upon the mucous membrane of the larynx in laryngeal croup. In the greater branches the calibre of the canal is not completely occluded; the coagula are tubular; but in the smaller bronchi they form cylindrical plugs. Croupous bronchitis is seldom spread over the whole lung; generally it is partial, and confined to a small number of bronchi; but to this rule there are exceptions. I know of a young girl, of fifteen years of age, who for years has almost daily coughed up a complete cast of the left bronchial tree.

SYMPTOMS AND COURSE.—The small extent of bronchial croup, as well as the absence of the fever, causes the progress of this disease to be quite different from that of croupous inflammation in the larynx. Indeed, while the latter is an extremely acute disease, croupous inflammation of the bronchial mucous membrane is, in some cases, a chronic one, which drags on for months, and even years.

The patients generally suffer from moderate dyspnœa, and nothing save the pale countenance and a certain relaxation and sleepiness indicate that respiration is carried on incompletely, and that the blood is not entirely decarbonized. From time to time, convoluted masses are ejected, after painful and spasmodic coughing. These become disentangled in water, and then present regularly-formed casts of the bronchial ramifications, consisting of tree-like, repeatedly-forked coagula, generally covered with a little blood. Upon auscultation, we hear exquisite rhonchus sibilans, corresponding to the extent of the bronchial croup; or, as in my case, the respiratory murmur is wanting as long as the tubes are filled with exudation, and returns as soon as the membrane has been expectorated. From time to time the disease, which, as we have said, is usually chronic in its course, takes on exacerbations. These often commence with a chill, and are followed by fever. The process sometimes spreads throughout other regions; great dyspnœa ensues; breathing may become insufficient, and death may take place under the often-mentioned symptoms of insufficient respiration.

Diagnosis.—Croupous inflammation of the bronchial mucous membrane is easily distinguished from catarrhal inflammation by the expectoration of characteristic moulds of the bronchial tubes, consisting of coagulated fibrin. As the mucus from the finer bronchi may sometimes have sufficient tenacity to retain its form, in doubtful cases, we may employ the test afforded by the different reactions of fibrin and mucin with acetic acid, the fibrinous clot swelling up upon application of the dilute acid, the mucus shrinking and becoming firmer. In well-marked cases error is impossible.

Prognosis.—Although the disease seldom attains such magnitude as to endanger life, yet the prognosis as to complete recovery is unfavorable. Bronchial croup is an exceedingly obstinate disease, evincing a great tendency to relapse, and often becoming complicated at last by tuberculosis, unless, indeed, this disorder accompany it from the outset.

Treatment.—The treatment is to be conducted upon the principles laid down for the management of laryngeal croup. In one case (*Thierfelder*), iodide of potassium afforded very marked and brilliant service (ʒ ss daily), so that this treatment should be imitated. I have never observed any benefit resulting from its action in this disorder.

## CHAPTER III.

### SPASM OF THE BRONCHI—BRONCHIAL ASTHMA—NERVOUS OR CONVULSIVE ASTHMA.

Etiology.—Long before it had been proved that the bronchi possessed muscles, still longer before we had discovered that these muscles could be experimentally made to contract by irritation of the par vagum, or, still more readily, by irritation of the mucous membrane, a great number of forms of asthma had been described in the pathologies. True, the causes of the greater part of them have since been traced to material alterations in the parenchyma of the lung, especially to emphysema, organic disease of the heart, etc. Nevertheless, there still remains a certain number of cases of motor derangement of the par vagum, in which the bronchial muscles contract spasmodically, and the calibre of the bronchi becomes narrowed.

It has already been stated that hyperæmia and catarrh are often accompanied by contraction of the bronchial muscles, a reflex phenomenon proceeding from reaction of the sensory fibres of the par vagum upon its motor filaments, and furnishing one of the sources of dyspnœa in bronchial catarrh. The term nervous asthma, however, is not to be applied to cases of spasm of the bronchial muscles, due to structural change of its mucous membrane, but merely to those in which the point of irritation of the vagus is remote, whether at its root or in its continuity, and to the instances in which we are forced to suppose that some irritation of nerve-fibres other than those of the par vagum induces a morbid excitement of its motor chords by means of reflex action.

The etiology of nervous asthma is as obscure as is that of spasm of the glottis. Disease of the heart and emphysema have been spoken of as predisposing causes. It is, doubtful, however, whether, besides the dyspnœa which they occasion, these affections ever give rise to abnormal action of the bronchial muscles. There are but a very few instances in which the source of the malady can be traced to disease of central organs, or to pressure upon the par vagum by a tumor. In other instances, especially in uterine disorders, nervous asthma accompanies other nervous derangements. Finally, it may attack persons *idiopathically*, who, in other respects, seem to enjoy perfect health, and in whom, after death, none of the above-mentioned abnormities can be found.

The exciting causes of the paroxysms are, in the majority of instances, equally obscure. Some persons never suffer from asthma at their own abode, but are invariably attacked whenever they spend a night in certain localities. In other cases, the inhalation of the dust from certain plants, especially ipecacuanha, will occasion a seizure. Finally, mental perturbation, sexual excess, inflation of the bowels (the laity ascribe a great deal to " bloating "), have a somewhat questionable claim to rank among the exciting causes of bronchial asthma.

The attacks of violent dyspnœa, accompanied by suppression of urine, which not unfrequently occur in the course of Bright's disease, and sometimes also in disease of the urinary passages, have been called urinary asthma. I, too, used formerly to ascribe them to poisoning of the blood by the constituents of the urine, or by the results of decomposition of these constituents. Recent observations of mine, however, which agree with those of *Bamberger*, seem to show that the dyspnœa depends upon œdema of the lung. During the last few years, as long as the dyspnœa lasted in such cases, I have always been able, upon auscultation, to find moist *râles*, which subsided together with the dyspnœa, upon the ejection of large quantities of watery sputa by means of violent vomiting or coughing. It would thus appear that urinous asthma is in some degree analogous to many cases of so-called uræmic convulsions. (See article, Bright's disease.)

Anatomical Appearances.—As we have seen, it is only in the rarest instances that we are able to find structural changes in the cadaver, to which the symptoms of bronchial asthma can be attributed without dispute. Indeed, in order to warrant a diagnosis of pure bronchial asthma, the bronchial mucous membrane should appear healthy, nor should any other cause for the dyspnœa be discoverable at the autopsy.

SYMPTOMS AND COURSE.—Bronchial asthma, like other nervous diseases, has a typical course, in which paroxysms alternate with intervals of exemption, although its type is seldom a regular one. Asthmatic attacks sometimes follow one another with short pauses, for a while, and then subside, often, not again to return for months, or even years.

Should the paroxysm come on during sleep, the slumber becomes restless, and the as yet unrecognized sensation of dyspnœa gives rise to the most frightful dreams. When the sufferer awakes, he has, as *Romberg* aptly describes it, "great desire to draw a deep breath, but feels that the air does not penetrate into the chest beyond a certain point. Hissing, whistling, purring noises are audible, both upon inspiration and expiration, which are perceptible to the patient himself, and may often be heard even at some distance. The embarrassment increases; the respiratory muscles, and even their auxiliaries, are brought into action; the alæ nasi work; the contour of the sterno-kleido-mastoidei muscles stands out distinctly; the head is thrown back, and the arms are braced, so as to expand the chest—but in vain. The vesicular murmur ceases, is replaced in some parts by hissing, which suddenly comes and goes, while the inspiratory sounds of the larynx and air-passages continue, not only undisturbed, but even are louder and stronger. Terror is depicted upon the countenance; the eyes are widely opened; cold sweat bedews the forehead; the complexion is pale, the impulse of the heart violent, unequal, irregular; the radial pulse is small and weak; the temperature of the hands and cheeks is depressed. Such an attack, after lasting for a quarter of an hour, or perhaps several hours with brief remissions, either ceases suddenly, the air rushing forcibly into the occluded bronchi and air-cells, causing puerile breathing, or a cessation comes about gradually, accompanied by eructations, yawning, more rarely coughing, with an increased secretion from the bronchial mucous membrane, which continues for some time."

The latter symptom of this truly classical picture finds a complete analogue in the course of a neuralgia of the frontal nerve. As in the one case the attack often ends in hyperæmia and swelling of the conjunctiva, with increased mucous and lachrymal secretion, in the other the neurosis of the vagus terminates with hyperæmia, and augmented secretion of the mucous membrane of the bronchi.

DIAGNOSIS.—If we form a distinct conception of nervous asthma, distinguishing between it and the spasmodic muscular contractions arising as reflex symptoms in bronchial catarrh, its diagnosis is easy. The severity of the paroxysms, the free intervals, the absence of symptoms of disease of the mucous membranes, keep us from error.

PROGNOSIS.—The prognosis as to life is better than the appearances would lead one to fear. Although the sufferer, often as he may have safely undergone an attack, fears that he will not live through the next, yet in the disease itself there lies a remedy to allay danger as soon as it arises. The moment that signs of carbonic-acid poisoning develop, the spasmodically contracted bronchial muscles are relaxed, as are also the muscles of the entire body, upon which the poisoning undoubtedly has a paralyzing effect.

TREATMENT.—Where chronic uterine disease is the cause of nervous asthma, the causal indication demands measures corresponding to the hysterical nature of the attack. In general, we are unable to meet the causal indication, the cause itself being unknown.

The indications for treatment of the disease itself are, first, to arrest, shorten, or mitigate the paroxysms; and, second, to take measures for the prevention of their recurrence.

To attain the first object, before all else the patient must be freed from all tight clothing, and his apartment supplied with pure, dry, warm air. The most efficacious of all dietetic remedies (in the widest sense of the word) is the *respiration of compressed air*, but it is rarely that a patient has the opportunity to prove its value during an attack. Very good results have been observed after a long abode in an institution provided with a well-contrived apparatus for this purpose. A gentleman of my acquaintance, for many years a sufferer from asthma, has had an apparatus constructed in his own house, within which to undergo his attacks in greater comfort. The great relief which asthmatic subjects experience upon inhaling compressed air is easily accounted for. The greater the pressure upon the gas which we respire, so much the more of it is taken into the blood; hence a moderate degree of obstruction to respiration may be compensated for by augmentation of pressure upon the gas respired. It is remarkable that this (in my opinion) principal cause for the efficacy of the inhalation of compressed air, the great influence of which upon the inspiratory act has been proved by the experiments of *Hoppe-Seyler*, is hardly referred to in his writings upon this subject.

The ancient and renowned recipe of a cup or two of strong Mocha coffee (two ounces to the cup), and the exhibition of small pieces of "water-ice," as recommended by *Romberg*, stand intermediate between the dietetic and medicinal remedies. In some cases either one or other of these articles will afford considerable relief; in others they fail entirely; and it is impossible to predetermine which of these results will occur. Among the medicines in the narrower sense of the word, the narcotics justly stand highest in repute. When the diagnosis is certain, doses of opium or of morphine may be ordered boldly. Where their internal administration fails, the experiment of a subcutaneous injection of a solution of morphine may be made. My own experience is not in favor of the tincture of lobelia inflata, so famed in treatment of asthma (gtt. x—xxx every 15—30 minutes). Nor have I observed much of the reputed benefit derived from smoking a pipe containing twelve or fifteen grains of stramonium-leaf, mixed with tobacco or sage-leaves, or from the use of the stramonium cigars of the shops, to say nothing of the fact that, in most instances, such procedures result in distressing headache. In a few instances, inhalation of chloroform vapor affords marked, but, as a rule, only temporary relief. The experiment of burning saltpetre-paper (blotting-paper soaked in a saturated solution of saltpetre and then dried) may always be made, although the vapors thus generated are very unpleasant to many asthmatic patients, and are of no benefit to them. In severe attacks we may administer an emetic, which often proves of great use. When the paroxysm threatens to prolong itself, instead of the emetic we may exhibit tartrate of antimony, or ipecacuanha, in nauseant doses. (*Köhler* especially recommends an infusion of ipecac, gr. v—vi to ℥ j, with extract pulsatilla ℈ ss.) This internal medication may be combined to advantage with cutaneous stimulation, such as friction upon the chest with warm turpentine, and the application of sinapisms to the wrists and calves of the legs, warm hand and foot baths. The patients, who feel as if in the agony of death, beg incessantly that "something more" be done to alleviate their sufferings.

*In order to avert future attacks*, we should, first of all, caution the patient to avoid exposure to any irritants which, according to his experience, have been the cause of previous paroxysms. Such instructions must be as exact and comprehensive as possible, no matter how odd the supposed source of the attacks may seem, and even although the connection between the cause and effect be altogether unintelligible. For instance, if the attack comes on whenever the patient sleeps in a dark or close chamber, he must always have a light burning, and leave his doors open. It is desirable that all asthmatic persons should reside in a pure, dry atmosphere; that they should avoid dusty, smoky, and windy localities, and that they should not sleep too long.

Among medicaments especially in repute for the prevention of new paroxysms, and for the radical cure of asthma, quinine stands first. The shorter and more regular the intervals of the attacks, so much the more is to be expected from this drug. It is unsuitable when the pauses between the seizures are very long or irregular in their occurrence. In such cases we must have recourse to other remedies from the list of the so-called *nervines*. Unfortunately, the mode in which this class of medicines modifies the function and nutrition of the nerves is very obscure, and the indication for the selection of one or other of them is so indefinite that we are reduced to a blind empiricism. (We must not, however, on this account, abandon this disease to itself any more than epilepsy, St. Vitus's dance, or other nervous affection.) As a rule, the metallic nervines (carbonate of iron, gr. v—x in die; zinci oxid. gr. ij—iv; argenti. nitrat. gr. $\frac{1}{6}$—$\frac{1}{8}$ in die; arsenic in form of Fowler's solution, gtt. iij—v in die) are preferred before tincture of valerian, assafœtida, castor, or camphor.

## CHAPTER IV.

### SPASM OF THE RESPIRATORY MUSCLES.

*Wintrich* and *Bamberger* have recently described highly-interesting cases of asthma, in which the dyspnœa is due to spasm of the diaphragm, and not to convulsive contraction of the bronchial tubes. That such a tonic cramp of the diaphragm must be highly embarrassing to the act of respiration is very manifest.

In this form of asthma, whose pathogeny and etiology are as obscure as are those of bronchial asthma, the loud, sibilant rhonchi, so characteristic of the latter, are wanting. The hinderance to respiration is distinctly limited to the expiratory act. During the latter the patient forcibly contracts the abdominal muscles, so as to overcome and force up the diaphragm, which has become fixed in the expiratory attitude by the tonic spasm. The belly becomes as hard as a board, and the contour of the abdominal muscles, especially that of the recti, becomes distinctly prominent. In consequence of the violent compression of the abdominal contents, the urine is passed involuntarily, and sometimes also the fæces. The patients become extremely cyanotic. Expiration requires two or three times as long as inspiration. The number of breaths in a minute falls as low as ten or twelve. Inspiration is proportionately short, and does not cause prominence of the epigastrium; the upper part of the thorax alone undergoing a moderate enlargement, while the lower is drawn in toward the spinal column. Percussion after extreme expiration also shows abnormal depression of the diaphragm. The clear percussion sound of the lung extends one or two inches too far downward, and the heart-shock and cardiac dulness are also displaced downward a distance of one or two intercostal spaces. Upon auscultation during the paroxysm, the respiratory murmur is inaudible.

The above symptoms require no explanation, as they are the immediate effects of tonic spasm of the diaphragm. Should the attack last long, consciousness fails, partially in consequence of disturbance of the cerebral circulation, and partially owing to the incompleteness of respiration, whereby the blood becomes overcharged with carbonic acid; the cyanosis becomes extreme; the pulse grows small, the skin cool, and in severe cases death may take place during the seizure. In more fortunate cases, after a longer or shorter duration, the spasm subsides gradually, the cyanosis fades, the dyspnœa ceases, the abatement, however being unaccompanied by any cough or expectoration, such as usually arises at the end of a fit of bronchial asthma.

In the cases reported by *Bamberger* the patient derived relief chiefly from use of the cold douche, inhalation of chloroform, and subcutaneous injection of morphia; but he eventually succumbed to the disease.

*Bamberger* is of the opinion that some forms of asthenia may proceed from spasm of other muscles of inspiration, and possibly, too, from spasm of the expiratory muscles.

## CHAPTER V.

### WHOOPING-COUGH.—STICK-HUSTEN.—COQUELUCHE.

Etiology.—According to our views, whooping-cough is a catarrh of the respiratory mucous membrane, distinguishable, however, from other catarrhs of this membrane, both by its mode of origin and by fits of spasmodic cough, which depend upon a peculiar hyperæsthesia of the air-passages.

The dependence of whooping-cough upon an unknown, but, undoubtedly specific cause, the frequent epidemic appearance of the disease, its propagation by infection, the almost positive protection against the malady afforded by a previous attack, remind us in many respects of the origin and propagation of measles, scarlet-fever, small-pox, and other forms of the so-called acute infectious disorders, of which we shall hereafter treat in detail. The circumstance, however, that the sole result of the specific exciting cause of whooping-cough is a local affection, while the rubeolar, scarlatinous, and typhous poisons involve the entire system, is a reason for distinguishing whooping-cough from these maladies. At the same time, it is not to be denied that even whooping-cough, as well as other epidemic contagious disorders, whose effects are merely local, is produced by the action of specific poison. Indeed, we regard this mode of origin not only as possible, but even as the probable one; but the nature and material of the infection in the disease in question must vary essentially from that of infectious diseases, in the narrower sense of the term, since their results are so very different. Inasmuch, however, as we have no definite knowledge regarding these morbid processes, and, since all the symptoms of this disease are easily traceable to local disorder of the respiratory mucous membrane, it will be more practical, on the whole, in spite of the contagiousness of whooping-cough, to treat of it as an affection of the respiratory organs.

In opposition to our views (namely, that whooping-cough is a catarrh of the respiratory mucous membrane, combined with intense hyperæsthesia of the air-passages), stand the opinions of physicians, who regard it either as a nervous affection of the par vagum, or else believe that it is a catarrh of the air-passages complicated by such a nervous affection. These views are based partly upon the spasmodic character of the coughing-fits, and partly upon the fact that the coughing-fits alternate with periods of complete exemption, a condition which is certainly suggestive of the typical course of the majority of nervous diseases. But coughing-spells of the utmost violence, and of the most pronounced spasmodic character, arise from reflex action in persons in perfect health, but whose respiratory mucous membrane has been exposed to irritation, such as contact with some sharp-cornered foreign body, grains of salt, or sugar, etc. In whooping-cough patients, coughing-fits, precisely similar, are provoked by slight causes, which might easily escape observation; but from such a fact we surely are not warranted in assuming the existence of a neurosis of the par vagum. The exalted susceptibility (the hyperæsthesia) of the diseased mucous membrane accounts fully for the ready occurrence and great violence of these *reflex* paroxysms of coughing. We shall have more to say regarding the typical course of whooping-cough when we come to discuss the symptoms.

Sporadic cases of whooping-cough are exceptional, a proof that the specific cause of the malady, if it develop spontaneously, usually attacks many persons, and that from a single individual the disease may spread to a great number.

Epidemics of whooping-cough occur most frequently during winter and spring, but do not die out upon the approach of warm weather. They often succeed epidemics of measles, or of scarlatina, and sometimes accompany them. The contagion seems to lie mainly in the secretions and exhalations of the diseased mucous membrane. The degree of its volatility and its other qualities are unknown.

Predisposition to whooping-cough is greatest in children, particularly in such as have attained their second year. It is an important fact that this predisposition is enhanced by any causes capable of producing catarrh, and still more so by the presence of any accidental irritation of the respiratory mucous membrane from cold or other cause. "Colds," slight but neglected catarrhs, as often furnish the exciting cause of an attack of whooping-cough as errors of diet, and catarrhal diarrhœas give rise to cholera.

Predisposition to whooping-cough always diminishes as age advances, and is extinguished almost without exception after one attack of the disease.

Anatomical Appearances.—Owing to the idea that whooping-cough is an affection of the nerves, special attention has been paid to the *post-mortem* condition of the par vagum and medulla oblongata. In a few cases it has been claimed that the neurilemma of the par vagum was relaxed or swollen, or that enlarged bronchial glands have been found so situated as to press upon the par vagum, or hyperæmia of the medulla oblongata and its membranes has been observed. Such observations, however, are but solitary; those, in which anatomical investigation fails to find derangement, either of nerves or central organs, are vastly more numerous.

There is no doubt but that, in whooping-cough, the respiratory mucous membrane undergoes some anatomical alteration, but to demonstrate its existence in the cadaver is a task of great difficulty. Owing to the richness in elastic fibres of the mucous membrane of the larger air-passages, any hyperæmia, which may have existed during life, disappears totally after death, leaving no trace behind. A moderate degree of swelling, relaxation, and infiltration of the mucous membrane may readily elude close observation, to say nothing of the *post-mortem* changes which may take place in these conditions also. However, the contrast between the symptoms during life and the appearances after death is no greater nor more puzzling in whooping-cough than it is in other catarrhal complaints. As this disorder, when simple and uncomplicated, very seldom ends fatally, we find almost always, in the cadaver of a whooping-cough patient, gross, palpable lesions, which are the results of the complication which has been the cause of death. In particular, we find that permanent inspiratory inflation (*Alveolarektasy*, see above) which even the best recent authorities still persist in confounding with emphysema, wide-spread atelektasis, catarrhal pneumonia in its several stages, and, far more rarely, croup of the air-passages or lungs, meningitis, or hydrocephalus. As these lesions do not depend upon the whooping-cough itself, but upon its complications, we refer to the appropriate chapters for further discussion upon these subjects.

Symptoms and Course.—It is customary to recognize three stages in whooping-cough: the catarrhal stage, the convulsive stage, and the stage of decline.

The *catarrhal stage*, in many instances, begins with violent catarrhal fever, with reddening of the conjunctiva, and great intolerance of light. The patient sneezes incessantly, and is tormented by a distressing cough. No one, without knowledge of the prevalence of an epidemic, could predict the approach of a whooping-cough at this stage of its development; nay, so far from receiving recognition as the incipient period of the disease, it is often mistaken for an intercurrent accident, in spite of its development into whooping-cough at a later period. In a few days the fever abates; the redness of the conjunctiva and the photophobia subside, together with the catarrh of the Schneiderian membrane. But the cough becomes more obstinate and persistent, and, at the end of each paroxysm, an astonishing quantity of a tenacious, viscid, transparent mucus fills the mouth and fauces. This peculiar, adhesive, copious secretion is pathognomonic of whooping-cough in its second stage, and establishes the diagnosis almost with certainty even at this period. Soon, however, the cough assumes a peculiar character; it is accompanied by violent reflex action of the muscles of the larynx, causing spasmodic closure of the glottis. From this point we date the beginning of the convulsive or whooping stage.

The coughing-fit begins with a long-drawn, clear, piping sound (produced as the air is slowly drawn into the constricted glottis). Then follows a series of short, rapidly-interrupted, expiratory coughs (the air, though vigorously expelled, being unable to force open the glottis for more than a moment at a time), and this, in turn, is succeeded by the crowing, long-drawn inspiratory act. Thus the "whoop" alternates with the cough, the latter finally becoming almost inaudible, until at last (though often not until after lapse of some minutes) the viscid secretion is brought up, and is removed intuitively by the mother, or else, what is more frequent still, is ejected by vomiting, together with some of the contents of the stomach. As shown elsewhere, spasmodic closure of the glottis during forced efforts at inspiration impedes the current of the jugulars, producing acute cyanosis. The patient grows deep red, or bluish, the fauces becomes swollen, the eyes shed tears, and seem as if about to burst from their sockets; the tongue looks thick and blue; the patient seems to be on the verge of suffocation. Bleeding from the nose and mouth and ears often takes place, and ruptures of vessels occur in the conjunctiva, which becomes infiltrated with blood so as to disfigure the patient for days and weeks. Hæmorrhage from the ear is caused by rupture of the membrana tympani.

The vomiting, which in bad cases empties the stomach of all its contents whenever the child coughs, is not always the only indication of the forcible compression of the abdomen. Sometimes we may observe involuntary evacuations of fæces and urine, although this is rare. At others, rupture or prolapsus ani is produced.

A sensation of tickling in the throat usually precedes each fit of coughing, the number of which, in the course of a day and night, may amount to twenty-four or upward. The children recognize these precursors of an attack with dread. They cling anxiously to their nurses, seek a support for the head, or begin to cry. After the paroxysm, they remain awhile exhausted, and suffering from pain along the insertion of the abdominal muscles. Soon, however, they recover, begin to play, or to eat with good appetite, until a new fit interrupts their comfort.

It is upon these alternations of symptoms that the theory is based that whooping-cough is a neurosis of the par-vagum nerve. Although laughing, crying, loud speaking, insignificant acts of deglutition, often give rise to the seizures, yet we cannot deny that they are not always the result of demonstrable external provocation. Indeed, we must admit that the paroxysms often increase in frequency during the night, although the room has not grown colder. It is difficult to assign a reason for this circumstance. If, however, we question an observing mother, or watch a sleeping child ourselves, until the whooping-cough awakens it, we may satisfy ourselves that the fit is preceded by a slight and very brief, although perceptible, laryngeal rattle, and if we look into the child's throat, as soon as he begins to cough, the pharynx will be found filled with that tough mucus the accumulation of which provokes the attack, and the ejection of which ends it. The secretion once discharged, some time elapses before a fresh collection of it produces a new coughing-fit, and thus the semblance of intermission is given to the apparently rhythmical march of the disease. Every coughing-spell is a new source of irritation to the mucous membrane of the larynx. The more violent it has been, so much the more rapidly does new secretion form, and so much the sooner is the next paroxysm to be expected.

The convulsive stage having lasted three or four weeks, or in other cases as many months, the *stadium criticum seu decrementi*, or stage of decline, gradually sets in. The catarrhal secretion loses its tenacious, transparent quality, becomes more liquid, yellow, and opaque. The sputa cruda become sputa cocta. Here, too, the altered secretion affords evidence that the hyperæmia and irritability of the mucous membranes are subsiding. The paroxysms are no longer provoked by slight external irritants; and the secretion becoming more easy of ejection, and the reflex symptoms growing milder, as irritability of the mucous membranes decreases, the fits themselves are shortened. The vomiting, which formerly closed the seizure, ceases to occur, excepting when, the larynx chancing to encounter some irritant of unwonted activity, a coughing-spell of the ancient violence is provoked. Relapses are extraordinarily apt to occur, if the child be not carefully protected from all pernicious influences. The mucous membrane remains extremely sensitive for months, although the disease be extinct and the child be recovered. Every slight catarrh is attended by spasmodic stricture of the glottis, upon coughing, and recalls to mind the old affection from which it has long been free.

Physical examination made during the interval affords no characteristic evidence. Percussion is normal, and auscultation reveals signs of catarrh. Other changes depend upon the complications. During the paroxysm, according to *Wintrich*, while the forced expiratory efforts continue, the percussion sound is shorter, feebler, and duller. This is probably due to the fact that, during the act in question, the air within the lungs undergoes considerable compression, and is thus made to press so heavily upon the pulmonary tissue and the thoracic walls that the latter are brought to so high a degree of tension that they cannot be set into vibration by percussion. Upon auscultation, no respiratory murmur can be heard during the protracted, sonorous inspiration. During the interrupted expiratory act likewise, although the vibration of the chest is communicated to the ear, no murmurs are distinctly audible.

Whooping-cough terminates in recovery in a large majority of cases. According to popular belief, such an event cannot occur before the eighteenth or twentieth week, a prejudice shameful for a physician, and highly dangerous to the laity, whom it betrays into a stupid *laisser aller*, and into all sorts of heedlessness. Under judicious treatment and systematic nursing, it is almost always possible to bring the disease to a close in from four to six weeks.

In other cases, which are by no means rare, the malady terminates in an incomplete recovery; umbilical and inguinal hernia result, and, what is still more common, that attenuation of the lung-substance and dilatation of the air-vesicles, hereafter to be described as emphysema. It is owing to the latter sequel of whooping-cough that many children, after having the disease, remain short of breath for the rest of their lives. The assertion, so often made, that whooping-cough may leave tubercles behind it, is to be accepted with some reserve. So long as the terms phthisis and tuberculosis were synonymous, such a statement was quite allowable. There is no doubt that a great number of children who have suffered from whooping-cough perish, sooner or later, from consumption of the lungs; but it is with comparative rarity that the form of consumption of which they die is the tuberculous form, due to the development and subsequent destruction of miliary nodules in the lungs. Most of the children, in whom the signs of phthisis appear, a few weeks or months after their having whooping-cough, are suffering from chronic catarrhal pneumonia, with cheesy metamorphosis, and subsequent destruction of the inflamed lung-substance. In such cases, catarrhal pneumonia, which has complicated the original disease, instead of undergoing resolution, results in the condition above described—a form of phthisis much more common than tubercular phthisis.

When a case terminates fatally it is almost always in consequence of complications, some of which consist merely in abnormal extension of the disorder, or in the effects of its intensity. If any of the bronchi become permanently occluded by the catarrh, so that no more air can penetrate to the vesicles in which they terminate, that which they already contain becomes absorbed, the vesicles collapse, their walls touch, and atelectasis is established. If the catarrh spread from the bronchioles into the air-cells, a catarrhal pneumonia is set up. We have already stated that nearly all fatal cases show traces of these complications. It should always awaken suspicion if the coughing-fits begin to lose their characteristic features, if an additional short, dry cough arise, if the patient grow short of breath, if the palms of the hands burn, if the sleep be troubled, the general health be disturbed, or other febrile symptoms appear. In all such cases we must repeatedly and carefully search for the signs of capillary bronchitis, of atelectasis, or of catarrhal pneumonia. Croup of the larynx or pulmonary vesicles is far easier of recognition, partly on account of the acuteness and violence of its initial symptoms, and partly because its signs are so obvious and unmistakable. This complication, however, is rare.

The cerebral symptoms which sometimes appear in whooping-cough scarcely ever arise from apoplexy, meningitis, or hydrocephalus. The vessels of the brain, if normally nourished, are not apt to tear, even though subjected to very severe pressure from within; moreover, diseases like meningitis and acute hydrocephalus are not to be induced by a simple and transitory hyperæmia. The convulsions, etc., which take place during whooping-cough, and which seldom endanger life, proceed either from a temporary venous obstruction with œdematous infiltration of the brain, followed by arterial anæmia, or else they are eclamptic seizures, caused by reflex action.

Diagnosis.—As the coughing-fits in this disease are, in my opinion, undistinguishable from those of any severe laryngo-bronchial catarrh arising from cold, or from the cough produced by the entrance of a foreign body into the larynx, or that which may be provoked in an extremely susceptible subject (such as an hysterical woman) by the most trifling irritation—indeed, as the cough owes its character to physiological processes, precisely like those which occur in the other varieties of cough—we deem it idle to talk of a differential diagnosis of the paroxysms. The epidemic appearance of the disease, its almost exclusive occurrence among children, its obstinacy, and long duration will prevent error. In infants at the breast, and very little children, the peculiar cough and reflex symptoms of the glottic muscles are sometimes absent, and, unless the expectoration of copious tenacious secretion and the prevalence of an epidemic of whooping-cough be borne in mind, the true nature of the affection may be overlooked.

Prognosis.—One must be aware how rarely children become apoplectic, or die in a whooping-cough fit, ere one can witness a paroxysm without anxiety. Experience accordingly teaches that the mothers, who at first are dreadfully alarmed, by-and-by grow only too careless, and at last, without further solicitude or attention, calmly await the advent of the twentieth week, when, as they think, the disease is to subside. Children who are but a month or two old encounter the greatest danger, not because "their foramen ovales is as yet unclosed," "and a commingling of blood from the two sides of the heart threatens to cause acute cyanosis," but because of obstacles to which they so often succumb in other bronchial catarrhs, the facility with which their little bronchi become occluded, imperilling life by atelectasis, or by deficient oxygenation without atelectasis. The danger arising from these complications, as well as that occasioned by the sequelæ, has already been treated of in detail.

TREATMENT—*Prophylaxis.*—As whooping-cough scarcely ever occurs except as an epidemic, and as it is often infectious, prophylaxis demands, where circumstances permit, an avoidance of places where the epidemic prevails, and separation of the sick from the well, particularly from those small, weak, scrofulous children to whom the disease would bring great danger.

As, moreover, the predisposition is augmented by catarrh, and by every irritant which can give rise to catarrh, it is well, in seasons of epidemic whooping-cough, to protect children with the utmost care from taking cold, and to treat the most simple catarrh with the same solicitude and precaution with which, in cholera-times, we put patients upon a rigid diet for every trifling diarrhœa, and look upon it as a dangerous illness. During epidemics I have kept children continuously confined in one room from the moment that any slight unsuspicious cough arose, and kept up a uniform temperature day and night for several weeks in succession, and have frequently seen children remain free from whooping-cough, while it developed among their relatives who had been attacked in similar manner, but who had been less carefully nursed. The *indicatio causalis* cannot be met, as we are not able to neutralize nor to remove the prevalent epidemic influence which excites the disease.

The happy effects which whooping-cough patients often obtain by change of abode are perhaps to be accounted for by the exemption from continuous or repeated exposure to the exciting cause of the disease, which they thus acquire. Whenever an extensive epidemic prevails about the dwelling of a patient of mine, I am in the habit of advising a temporary migration to some region free from the disorder.

The *indicatio morbi* calls for the same general treatment which we have recommended for other laryngeal and bronchial catarrhs. *Recognition of the fact, that in whooping-cough we have a catarrh to deal* *with*, should deter us from a useless and hazardous search for specific remedies. Unfortunately, the principles of treatment naturally deducible from such knowledge are grossly violated by many physicians. Children are again and again submitted to hurtful exposure, calculated to aggravate the catarrh, because the doctor expects to cure it by the administration of preparations of copper and zinc, nitrate of silver, valerian, assafœtida, castor, or other reputed specific, against whooping-cough; nay, cantharides, phosphorus, arsenic, and even *dried vaccine crusts*, pass for remedies for this disorder in the minds of some men. We cannot even ascribe any special curative influence to belladonna, a drug which has acquired great reputation, although we have used it extensively in the treatment of whooping-cough. (See below.)

Since, then, the complaint is to be treated upon principles laid down for the treatment of other catarrhs, we need not expect great results from the use of medicaments. We have already called attention to the very questionable virtues of sal-ammoniac, the antimonials, and other anti-catarrhal "remedies," and hence are decidedly averse to their employment in the therapeusis of whooping-cough. On the other hand, we attach great value to well-managed treatment by sweating, the efficacy of which, in both recent and inveterate catarrhs of other origin, I have already insisted upon so urgently. *Oppolzer* believes it possible to bring the malady to a close in a few weeks, by carefully and continuously maintaining a uniform temperature in the sick-room. Although such a statement may be a somewhat exaggerated one, yet I have long since adopted the practice, and in many instances strictly and perseveringly carried it out with the most gratifying results. When the disease is recent, I put the child to bed, and keep it in a slight but continual perspiration. Very young children must not lie in a cradle, but should remain in bed alongside of their nurse or mother, where they soon get into perspiration. Besides, they should wear a woollen sock round the throat, and wear flannel next the skin, upon the chest. Throughout the warm season of the year, the patients may be kept in the open air; but we should insist upon their returning to their apartments ere the cool of the evening set in. As a drink, give Seltzer water, either warm or mixed with hot milk; in short, treat the catarrh as if it proceeded from cold, or had any other non-specific origin. Treatment by inhalation of pulverized medicaments may possibly prove beneficial; but we have not as yet a sufficient number of observations to establish the fact. The symptomatic indication, first of all, calls for means of abbreviating the paroxysms of coughing, and of diminishing their frequency. It cannot be ignored that the coughing-fits contribute materially toward keeping up the irritation of the mucous membrane. The longer and more violent the last attack, so much the sooner may we expect the next one; hence, if we can succeed in rendering the seizures milder and less numerous, we not only produce results which are palliative but radical, by enabling the disease the sooner to cease spontaneously. Reduction of the number, severity, and duration of the paroxysms is as important for the cure of whooping-cough, as is prohibition of loud talking in the treatment of obstinate hoarseness.

For this purpose I cannot sufficiently urge that the parents, if intelligent and persevering, be directed to make the child cease from coughing as soon as possible, and, if necessary, even to enforce this harsh demand with wholesome sternness and severity, as soon as the mucous accumulation is discharged. A portion only of the coughing is involuntary. By an exercise of firmness, a child may withstand the remaining inclination to cough. The mother, however, must never tire of warning, admonishing, and, if need be, threatening, though no immediate benefit become apparent, even after lapse of days; for this mental dietetic must be maintained for weeks and weeks.

I have heard the assertion made by the wife of a Prussian general, a most determined woman, but an equally tender mother, that whooping-cough was only curable by the rod. Such a statement as this, and the advice to tell a child to stop coughing, and even to compel it to resist the cough as much as it can, have excited objection, here and there, and have even given rise to some virtuous indignation. Notwithstanding this, however, further personal experience and the approval of other judicious practitioners induce me emphatically to reiterate my counsel. It is of course inapplicable where the parents are stupid or rude; but the physiological law is well known, that violent reflex symptoms are controllable by the will. Perhaps the fact that adults are better able to resist the inclination to cough, and do not abandon themselves so completely to the irritation, explains why exposure to the same causes will, it is true, bring on catarrh, but rarely a catarrh of the intensity and persistence observable in the whooping-cough of children.

This treatment is materially aided, if, as soon as the child perceives a fit to be coming on, or when the fine *râles* in the larynx above described give notice of its approach, a moderate dose of carbonate of soda or of potash be promptly administered. As the alkaline carbonates reduce the viscidity of mucus, and as the secretion collected about the epiglottis, if deprived of its tenacity, is the more easily expelled, and finally, since with the ejection of the mucus the paroxysm usually terminates, theory and practice concur with tolerable harmony in approving this measure. There is a mixture much employed in whooping-cough (℞ Coccinellæ, gr. xij; potas. carb. ℈j; aquæ dest. ℥iij; syrup. simpl. ℥j.—M. S. a teaspoonful when the attack threatens), the effect of which in shortening the fits of coughing is often surprising, but which hardly owes its virtue to the cochineal. Half a tumbler of soda and water, drunk occasionally, would probably produce about the same result. This mixture should not be given as a matter of routine, "a teaspoonful every two hours," or the like, but only when there is a collection of viscid mucus in the throat, and when a paroxysm is approaching. Prescribed in the latter way, parents are glad to use it, as they soon find out that it renders the attacks milder and shorter, or, as they say, "loosens the cough."

Narcotics have also been prescribed to shorten the coughing-fits and reduce their number, and there is scarcely an article of this large class of drugs which has not been recommended in whooping-cough, and even vaunted as a specific. This is especially the case with belladonna. We have already expressed ourselves in favor of a bolder use of narcotics in treatment of catarrhs, and hence approve of their use in whooping-cough, which we regard merely as a catarrh complicated with an especially intense hyperæsthesia of the air-passages; but we cannot admit that any narcotic whatever (belladonna included) has any specific action against this disease. If they render the course of whooping-cough briefer, they act by mitigating and lessening the frequence of the paroxysms, which are perpetuating the irritation. Since, however, as as a general rule, narcotics are not well borne by children, being apt to cause hyperæmia of the brain, we should restrict their use to those cases in which danger from the disease outweighs danger from the remedy. Should the treatment, given above, prove ineffective, should the child empty its stomach with every paroxysm, should his nutritive condition begin to suffer, from constant vomiting and from sleepless nights, should convulsions or signs of actual suffocation occur during the seizures, the administration of narcotics is indicated. Belladonna enjoys this advantage over opium, that, in the condition of the pupil, we have an index for regulation of the dose. To children, between the ages of two and four years, we may give the eighth of a grain of the drug, night and morning, gradually increasing the dose up to half a grain, or until the pupil begins to dilate. *Trousseau* preferred not to divide the daily dose, but gave it all at once, and he thought it useless to push the dose to the point of incipient narcotism. For older children, to whom the exhibition of opium is less hazardous, we may prescribe small doses of morphia (gr. $\frac{1}{12}$—$\frac{1}{8}$ in die), dissolve in some liquid like the aqua amygdalarum, so much lauded by *West*, or we may give a few drops of laudanum.

Another and very important indication is, to prevent the secretion from accumulating in the minuter bronchial tubes. We have already pointed out that such obstructions are a source of danger in themselves, being the cause of atelectasis. When we come to discuss the subject of catarrhal pneumonia, we shall show that this disorder depends not merely upon a propagation of inflammation from the bronchioles into the air-vesicles, but that obstruction of the bronchi and the consequent collapse of the lung also play an important part in its etiology. Emetics, therefore, very justly, are highly thought of in the treatment of whooping-cough, although we cannot approve of their administration every second or third day, without any particular indication, which practice is common enough. The smaller the child, the narrower its bronchi, so much the more danger is there of their obstruction, and so much the more sedulously must we watch them. If, immediately after the cough, there still remain audible *râles* in the chest, if the child grow short of breath, or should the respiration become enfeebled throughout a part of the thorax, do not dally until signs of imperfect decarbonization of the blood set in, but give an emetic forthwith, and repeat it whenever the symptoms recur. We have already explained why it is that emetics unfortunately do not always produce the desired effect.

Finally, especially in the third stage, when impoverishment of the blood and general exhaustion furnish the most urgent symptoms, the free administration of meat-diet, eggs, wine, and the ferruginous preparations is indicated.

# SECTION III.

# *DISEASES OF THE PARENCHYMA OF THE LUNG.*

## CHAPTER I.

### HYPERTROPHY OF THE LUNG.

IN by far the greater number of cases in which the lungs appear to be enlarged, their tissue, instead of being hypertrophied, is atrophied and wasted—or rarefied, to use a customary expression. Under the title of emphysema, we shall treat more fully upon this form of enlargement of the lungs in the third chapter.

*Rokitansky* describes an enlargement of the lung with simultaneous increase of tissue, as true pulmonary hypertrophy. This is said to occur as a vicarious development of one lung, when the other has been totally destroyed. Here the walls of the vesicles are said to be thicker and more voluminous, their capillaries multiplied, and their tissue more resisting, while the vesicles themselves are dilated.

A second form of hypertrophy of the lung, which *Skoda* describes, and *Virchow* calls pigmentary induration, consists in an increased volume of the pulmonary tissue, at the expense of the air-vesicles. We notice it in severe chronic hyperæmia of the lung, particularly in valvular disease of the mitral valve, and in hypertrophy of the right side of the heart. Here, too, the walls of the vesicles are thickened and their elements multiplied, so that, as the lung has not grown larger, the vesicles must seem smaller, and the tissues closer and more resistant. The color of the tissue is darker and browner, and we notice numerous blackish specks in it. This coloring is due to capillary hæmorrhages in the tissues, resulting from the intense engorgement, the pigment which proceeds from transformed hæmatin admitting of demonstration under the microscope in the form of brownish and blackish granules, in the interstitial tissues and in the epithelium of the air-vesicles.

Either form of hypertrophy of the lung may be suspected to exist during life, but neither ever admits of any certain diagnosis.

## CHAPTER II.

### ATROPHY OF THE LUNG.—EMPHYSEMA SENILE.

By atrophy of the lung we mean that attenuation, thinning, and gradual wasting of the vesicular walls, which results from imperfect nutrition of the lung. We most commonly see this wasting of the pulmonary tissue in old age, when it is accompanied by atrophy of other organs, and by general marasmus. Sometimes the atrophy of the lung occurs sooner and more completely than involution of the rest of the body, and in these cases the symptoms are more decided. Through wasting of their septa, several, and sometimes a great many, of the air-vesicles coalesce, and large gaps form, so that, in an aggravated case, the entire lung forms a coarse network. The relaxed, bloodless, dry tissue feels soft as down to the touch; it is heavily loaded with pigment, and is sometimes uniformly black. This deposit of pigment is not a result of extravasation of blood, but is due to pigmentary metamorphosis of the contents of obliterated capillaries. These nutritive derangements of the pulmonary tissue are essentially like those to be described in the next chapter, as characteristic of vesicular emphysema of the lung, so that the title of senile emphysema is not altogether inappropriate to the disease in question. In senile atrophy, or senile emphysema, however, the volume of the lung is diminished, while in emphysema, in the stricter sense of the word, it is increased. This important difference, which furnishes a practical distinction between the two affections, depends upon the difference in the condition of the thorax. The size of the lung manifestly depends upon the capacity of the chest. Now, while in true emphysema the thorax is either in a state of chronic enlargement, or else in a condition of permanent inflation, in senile atrophy the chest is actually contracted, or else it is in a state of permanent expiratory collapse. The thorax of an old man is shortened by absorption of the intervertebral substance, and laterally compressed by his stooping attitude, and the atrophied muscles of inspiration are incapable of dilating it effectively. For a similar reason the heart and liver, which, in true emphysema, are extensively covered over by the lung, in senile atrophy lie in contact with a large part of the thoracic wall.

This state of the lungs and chest explains why old men become short of breath, why their blood is imperfectly decarbonized, and why they are so apt to look blue about the lips and cheeks. The shortness of breath and the venous state of the blood are due to the diminution of breathing-surface by loss of cell-walls, to a decrease in the number of capillaries, and, finally, to the incomplete manner in which inspiration is effected by the wasted respiratory muscles, and to the absence of that important aid to expiration, elasticity of the lung. The cyanotic symptoms occur because the right heart, having fewer efferent channels, owing to obliteration of so many pulmonary capillaries, is imperfectly emptied, thus causing venous engorgement of the aortic circulation. According to this explanation, the more unequally atrophy of other organs and general reduction of the volume of the blood keep step with the atrophy of the lungs, so much the more marked must the symptoms, above described, become.

As the thin, emaciated, thoracic walls and their flexible ribs are easily thrown into vibration, the percussion-sound is remarkably loud and full. The dulness over the heart and liver is increased. Upon auscultation, the greater the difference is between the capacity of the contracted bronchioles and that of the air-vesicles in which they terminate, so much the harsher is the vesicular murmur.

Treatment of senile atrophy of the lung is out of the question.

## CHAPTER III.

### EMPHYSEMA OF THE LUNG.

By emphysema of the lung is meant either a morbid enlargement of the pulmonary vesicles, arising mainly from the blending of several vesicles so as to form one great cyst—*emphysema vesiculare*—or else the escape of air into the subpleural and interstitial connective tissue—*emphysema interlobulare.*

Etiology.—The opinions of authorities as to the mode of origin of vesicular emphysema of the lungs differ materially. Four principal theories have been advanced upon the subject. According to the first, emphysema arises through immoderate or too protracted inflation of the air-cells, by forced and long-continued inspiration—theory of inspiration. According to the second, likewise, emphysema is the result of excessive mechanical distention of the vesicular walls, but is not produced by inspiration, but by forced expiration—theory of expiration. According to the third, its origin is not mechanical, but it arises from nutritive derangement of the lung-substance, which occurs independently of any strain or stretching. Finally, according to a fourth view, advanced by *Freund*, a morbid condition, namely a rigid enlargement of the thorax, constitutes the primary disease, to which dilatation of the air-cells is only secondary. We think it narrow-minded and erroneous to admit only one or other of these views as correct, and believe that we can prove that each of them is true in certain cases, while no single one of them will account for all forms of the disease. We adhere to the old and judicious classification into ***vicarious*** and ***substantive*** emphysema.

*Vicarious* emphysema arises, first, in all cases in which portions of the pulmonary substance become wasted, and shrink without corresponding reduction of the capacity of the thorax, by collapse of its wall. If the capacity of the chest remain constant, the size of the individual vesicles which contribute to fill it must, of course, depend upon their number. If a portion of them perish, thus reducing the bulk of that part of the lung to which they belong, either a vacuum must form in the chest, or else the remaining vesicles must dilate. Secondly, vicarious emphysema arises where *all of the vesicles do not participate alike* in filling out the additional space *formed in the thorax by inspiratory dilatation.* Normally, all the air-vesicles are inflated uniformly by atmospheric pressure during inspiratory dilatation of the chest. If, then, a part of the air-cells be filled up by exudation or serum, so that no more air can enter them, these cells will not dilate during inspiration, and hence the *remaining* and accessible ones must expand for them—*vicariously*—and undergo an abnormal degree of distension. Thus, in the bodies of all persons who have died of pneumonia or hypostatic engorgement, we find vicarious emphysema in the portions of lung spared by the original disease. So, too, extensive rigid adhesions between the pulmonary and costal pleura give rise to vicarious emphysema. Under normal conditions, the air-cells in the apices of the lungs, and those placed near the spinal column, although the portion of the thoracic wall adjacent to them scarcely takes any part in the inspiratory movement of the chest, expand equally with the cells in the more movable regions of the thorax, and which are situated near to the diaphragm, and to the anterior thoracic wall. Of course, for this to take place, the adjacent movable parts of the lung must yield and be pushed downward and forward. Where the costal and pulmonary pleura are firmly united, such yielding and displacement become impossible, and the vesicles at the apices and along the back-bone cannot fully perform their part in occupying the space created in the chest by its inspiratory expansion. Hence, other portions of the lung, especially the anterior and lower borders, must act for them, and are thus made to undergo an excessive and abnormal distention. Finally, chronic catarrh of the smaller bronchi is often followed by vicarious emphysema. If, in one part of the lung, the calibre of the finer tubes be so much narrowed by swelling, or by accumulation of mucus, that the air enters imperfectly, and with difficulty, into the corresponding vesicles, such part of the lung will not do its share in filling up the inspiratory expansion of the thorax, so that other parts of the lung, which are free from catarrh, must act for them, and become abnormally distended.

From the foregoing representation it is evident enough that the "inspiratory" theory is a correct one. If vicarious emphysema arise acutely during a pneumonia or hypostatic engorgement, the intervesicular septa are ruptured. On the other hand, if the affection develop slowly, as in a case of adherent pleura, or of tedious catarrh, the vesicular walls undergo a gradual atrophy, growing thinner and thinner, and becoming perforated, until at length several cells coalesce into one large cyst from destruction of their septa. We must decidedly oppose the widely-spread impression that the over-distended cell-walls simply lose their elasticity, without other change of texture (just as any over-stretched glove or caoutchouc-pipe remains permanently enlarged). That an emphysematous lung has lost its elasticity is true, but the above explanation is false. The loss of elasticity is owing solely to the rupture above mentioned, or to the gradual wasting of the elastic elements of the tissue.

*Substantive* emphysema—that is, a form of vesicular emphysema, wherein the enlargement of the pulmonary cells is a more primary and independent disease—likewise arises, in many instances, in consequence of immoderate and protracted inflation and stretching of the vesicular walls. *Laennec*, who first pointed out the mode of origin of substantive emphysema, conceived it to be as follows: If, in consequence of catarrhal swelling of the mucous membrane, or owing to viscidity of the bronchial secretion, an obstacle to the passage of air arise in the smaller bronchial tubes, such obstacle may be surmounted during inspiration, owing to the powerful muscular force by which this act is effected; while to expiration, which has fewer auxiliary means at hand, and which is effected mainly through the elasticity of the lung and of the thorax, and through the counter-pressure exerted by the intestines during inspiration, such obstacle may prove insuperable. Thus a portion of the air is retained in the vesicles. The next inspiratory act adds more, which, likewise, is unable to escape completely, so that the vesicles are continually becoming more and more over-filled and distended. Two main objections have been advanced against this theory: First, it is replied that the supposition is erroneous that the auxiliaries to inspiration exceed those of expiration—at all events, as regards forced expiration. This is true; yet we have already shown that it is chiefly the larger bronchi which are emptied by a forced expiratory effort, while it has very little action in promoting evacuation of the air-vesicles, especially if the bronchioles be obstructed. It is thus that we account for the common occurrence of permanent *vesicular ectasia* of capillary bronchitis. Secondly, it has been urged against the explanation of *Laennec*, that this hypothesis would be satisfactory if the enlargement of the pulmonary vesicles were no greater in emphysema than the enlargement which they normally undergo in healthy lungs at the height of the inspiratory act, but that it does not account for the abnormal and excessive distention of the air-vesicles of an emphysematous lung. This argument ought to have prevented the confusion which exists between permanent inspiratory ectasis and emphysema of the lung. Permanent inspiratory ectasis may subside completely; and, indeed, after removal of the obstacles to expiration, often does so subside. (These are the cases of so-called recovery of emphysema of the authors.) *But, if permanent inspiratory vesicular inflation be kept up for any great length of time, the continuous strain and expansion produce structural changes in the vesicular walls.* They atrophy, grow thin, become perforated, several vesicles blend into one larger cyst, and then emphysema really commences.

This account of the origin of *substantive* emphysema, from immoderate *inspiratory* expansion and stretching of the vesicular walls, is not applicable to all cases, however, and there are a good many instances in which we must give credit to the *expiratory theory.* It is easy to understand how emphysema may arise in the upper part of the lungs as a result of often-repeated forcible expiration with simultaneous contraction of the glottis. In severe paroxysms of coughing, such as occur in whooping-cough and chronic bronchial catarrh, the thorax is vigorously contracted, while, at the same time, the escape of air is impeded by constriction of the glottis. In straining, and in playing upon wind-instruments, the same process occurs. So, too, in lifting heavy weights, and in other severe bodily exertions, the air is compressed within the thorax, and only allowed to escape at intervals, and with a groaning or panting sound. In all these acts contraction of the chest is effected by vigorous upheaval of the diaphragm. The result is the expulsion of a strong current of air from the lower bronchi, the direction of which is obliquely upward, and, if the air be prevented from escaping through the larynx, a portion of it, in a compressed state, must be driven into the upper bronchi, whose direction is obliquely downward. By the centrifugal pressure exerted, by the air thus compressed, upon the vesicles of the upper lobes of the lung, and upon the adjacent thoracic wall, the latter become distended as far as it is possible for them to yield. In a vigorous man, whom I have had under observation, and in whom the pectoralis minor and a larger portion of the pectoralis major were absent, I have been able to see that, both in coughing and straining, so much air was forced into the upper part of the lung as to cause prominence of the upper intercostal spaces; and I have often noticed and pointed out the same phenomena, though in a lesser degree, in patients with thin muscles, and but little subcutaneous fat.

The occurrence of emphysema, in patients who have not been exposed to any of the causes hitherto mentioned, is rare, but the fact is certain. Hence, in a small number of cases, it would seem that the third of the above theories of the origin of emphysema is the correct one; according to which, wasting, perforation, and final disappearance of the vesicular septa are not always due to a mechanical process, but may proceed from nutritive disturbance, and from other causes at present unknown to us. Perhaps most cases of inherited emphysema belong to this class.

The occurrence of rigid dilatation of the thorax as a primary disease, causing permanent inspiratory expansion, strain, gradual atrophy of the cell-walls, and consequent emphysema, according to the theory of *Freund*, is certainly rare and exceptional. As we shall show by-and-by, in a large majority of instances, rigid dilatation of the chest and the nutritive disorder characteristic of emphysema occur together, and arise from the same causes. I cannot, however, deny having met with a few cases which seemed to support the theory of *Freund*—cases in which a marked rigid dilatation of the chest had occurred during the period of puberty, without any sign of emphysema, but in which extreme emphysema had developed a few years later.

*Interstitial emphysema* arises from an over-distention and rupture of the air-vesicles adjacent to the pleura and interlobular interstices, with escape of the air into the sub-pleural and interstitial connective tissue. This excessive distention takes place sometimes from very violent coughing, like that of whooping-cough, sometimes in cases where a large number of air-cells are prevented from taking part in occupation of the space created by inflation of the chest. The frequent, though somewhat inexplicable, appearance of interstitial emphysema after croup is, perhaps, most probably attributable to the bronchitis, which, according to *Bohn* and *Gerhardt*, constantly complicates laryngeal croup, and often causes partial collapse of the lung.

Predisposition toward emphysema is sometimes congenital. It often appears during childhood as a result of whooping-cough (which is almost exclusively a disease of that age), as well as in consequence of the frequent and tedious bronchial catarrhs to which scrofulous and rachitic children are so liable. The period of advanced middle life, however, furnishes the main portion of emphysematous patients, when the "catarrh sec" is most prevalent.

From the above it appears that the exciting causes of the malady are chronic inflammation and wasting of the lung, inflammation of the pleura, with extensive adhesions, chronic catarrh of the lesser bronchi, with contraction of their calibre; frequently-recurring coughing-fits, especially those of whooping-cough and of dry bronchial catarrh; the playing upon wind-instruments, and similar procedures; lifting heavy burdens, and severe corporeal exertions. In some cases the exciting causes are unknown.

Anatomical Appearances.—The almost universal statement, that, upon opening the chest, the emphysematous lungs bulge forcibly out of it, is incorrect, or at least inexact. This active projection of the lungs from the thorax, when opened, is not the effect of emphysema alone, and is only met with in cases where, besides the emphysema, there is a wide-spread obstruction of the smaller bronchi, whereby the escape of air from the chest is prevented, while, during the last breath of the dying patient, the respiratory muscles relax, and the thorax collapses. True, emphysema is very frequently complicated with capillary bronchitis, which is the chief cause of obstruction of the minuter bronchi and of the retention of air in the vesicles; and, we may add, that emphysema, by reducing the elasticity of the vesicular walls, and thus diminishing their expulsive power, favors the occurrence of permanent inspiratory expansion. A very small obstruction in the bronchi of an emphysematous lung suffices to hinder evacuation of the air-cells.

Although the forcible protrusion of emphysematous lungs through the opening in the chest is by no means of constant occurrence in emphysema, and although it is not due to the latter disease but to one of its complications, *yet incomplete retraction of the lung, when the thorax is opened, is constant and peculiar to this affection.* Even where there is no complicating contraction nor occlusion of the bronchioles (which would likewise prevent retraction of the lungs), emphysematous lungs, when removed from the chest, or when merely exposed, remain more inflated, and larger than healthy lungs. In order thoroughly to comprehend this condition, about which also very erroneous ideas have been propagated, we must understand why a healthy lung, upon dissection of the thorax, contracts to a certain extent, and then remains without escape of the rest of the air which is in the vesicles. When, by exposure of the lungs, upon autopsy, we allow the atmospheric pressure to act upon their exterior surface, all air would immediately escape from the vesicles, and their walls would collapse like those of an empty sack, were it not that their outlets open into those narrow, heavily and thickly-walled tubes, the bronchi. Owing to their weight, the bronchi soon collapse, and thus prevent further escape of air from the vesicles. A simple experiment will demonstrate this fact: If we remove a stomach and œsophagus from a body, and, after tying the pylorus, inflate them, only a portion of the air will escape from the stomach after we cease blowing, because the heavy walls of the œsophagus collapse and oppose its further egress. This would be still more marked, did not the elasticity of the air-cells, to some extent, counteract the resistance set up by the weight of the bronchial wall. The vesicular walls of an emphysematous lung are thinner and slighter than those of a healthy lung. Besides, they have lost a considerable part of their elasticity, so that the reason is evident why lungs thus diseased remain more puffed up and inflated, when exposed to the atmospheric pressure, than do healthy lungs. Since emphysematous lungs, as they do not contract, retain about the same volume when the chest is laid open as they did when it was closed, we may further understand why, in cases of wide-spread and especially of substantive emphysema, in which, during life, the dimensions of the lungs had been abnormally large, there should also be found an increase in their size, when exposed post mortem. This increase in the volume of the lungs, as we shall show by-and-by, is the result of an increase in the capacity of the thorax, arising in part from depression of the diaphragm, and in part from an elongation of the costal cartilages, and a peculiar direction of the ribs. If an emphysematous lung were to contract like a normal lung, this increase in size would disappear if the chest were cut into; actually, however, it persists even after the lungs have been removed from the body.

In the *circumscribed vicarious emphysema about indurated or atrophied parts of the parenchyma*, we find groups of tender vesicles, varying in size from that of a hemp-seed to that of a pea. They are tightly inflated, and always traversed by a delicate network, a remnant of the intervesicular septa. The seat of circumscribed vicarious emphysema is most frequently at the apex of the lungs.

When vicarious emphysema has arisen acutely, during a pneumonia or hypostatic engorgement, or chronically as in pleural adhesion, the affection is limited to the anterior and lower edge of the lungs. This will be readily understood, after the explanation of its pathogeny given above. On the other hand, where the disease is a result of closure of any of the bronchi, the seat of the cellular dilatation is not so constantly at the anterior and lower edges, and often occupies the apices and other regions. A characteristic of this variety is, that inflated emphysematous portions of lung exist alongside of collapsed and shrunken parts. Such lungs, when removed from the chest, often present a very irregular contour, which does not at all correspond with that of the thoracic cavity. The emphysematous parts are prominent, and may even appear almost bulbous or pedunculated, while the collapsed region is marked by deep depressions and rigid bands. If inflated, it of course assumes the shape of the cavity of the chest, the hard cords and the pedicles of the bulbous portions retract into the interior, and the projections and lobes created by the depressions crowd together.

If the substantive emphysema arise from forced expiration and constricted glottis, its seat is always in the upper lobes of the lung and particularly in the apices. Where it proceeds from protracted inspiratory strain, the vesicular distension is distributed more or less evenly throughout the whole organ, and it is a characteristic of this variety, when removed from the chest, that the lungs maintain their normal contour, in contrast to the variety of vicarious emphysema last described. The same conditions obtain, both with regard to the extent of the malady and to the maintenance of the natural shape of the lungs, in that form of emphysema which arises without mechanical origin, and also in the variety whose source lies in a primary ectasis of the thorax.

If the entire lungs or their upper portions be the seat of emphysema of much intensity, the anterior pulmonary walls are pushed far downward. The left lung often covers the pericardium completely; the right reaches down to the seventh rib; the heart, usually much enlarged, lies upon the diaphragm in a more horizontal direction. In previous editions of this work there are many erroneous statements with regard to the position of the heart in emphysema.

An emphysematous lung feels remarkably soft to the touch, like a pillow filled with down. Upon cutting it, a dull, creaking sound is barely perceptible. The inflated tissues of acute emphysema are of a strikingly bright-red and pale hue; in chronic cases, as a rule, they are exceedingly dark, with here and there irregularly-formed black spots, the produce of conversion into melanin of hematine contained in the former capillaries.

Even to a superficial glance, the vesicles are seen to be greatly enlarged, sometimes to the size of a pea or bean, and are of irregular shape. Upon closer examination (of a dried specimen especially) in acute cases, we find, traversing most of the large cysts, a few delicate bands, as vestiges of the lacerated interalveolar septa. In mild degrees of chronic emphysema, we find perforations of the septa, varying in size and number; in severer cases, the walls are reduced to the condition of solitary ridges. At first, the atrophy is limited to the septa between the vesicles of one infundibulum; but afterward the vesicles of one infundibulum blend with those of its neighbors, by wasting of the intervening walls; until at last, in the worst stages of the disease, a large part of the pulmonary tissue is converted into a coarse network. As many of the capillaries perish with the septa, the emphysematous tissue is remarkably bloodless and dry.

Interlobular emphysema forms small cysts, full of air, beneath the pleura, which causes the latter to seem as if lifted by froth. These air-bubbles may be displaced by pressure, thus furnishing an easy means of distinction between vesicular and interlobular emphysema. This displacement, however, cannot be produced uniformly in all directions, but only in lines which mark the boundaries of the lobuli. Single lobules are often enclosed, like islands, by a narrow border of minute vesicles. This fact alone suffices to refute the assertion of certain French authors, that, in vesicular emphysema, the air is not enclosed in the air-cells, but in their interstices. There is no intervesicular connective tissue in the lung, it exists only between the lobules. More rarely there are larger cysts by which the pleura is extensively separated from the lung, and it scarcely ever happens that the pleura itself at last gives way, allowing air to enter its cavity, or that the air beneath the pleura penetrates along the roots of the lungs into the areolar tissue of the mediastinum, and thence to that beneath the skin, producing emphysema of the subcutaneous cellular tissue.

SYMPTOMS AND COURSE.—Circumscribed, vicarious emphysema, in the vicinity of portions of withered and shrunken lung, cannot be recognized during life, and has more pathological and anatomical than clinical interest.

The more slowly a person has died, the more distinct the symptoms of extensive hypostasis have been, the more forcibly the thorax has been expanded in the death-agony, so much the more surely may we expect to find an *acute vicarious emphysema* of the anterior and lower pulmonary borders at the autopsy.

The symptoms of extensive vicarious emphysema and those of substantive emphysema are very similar to one another, as the most prominent characteristics depend upon a more or less advanced wasting of the intervesicular septa, a nutritive derangement common to either form.

By-and-by we shall briefly adduce the points upon which we base a differential diagnosis between vicarious and substantive emphysema. In many instances the two forms cannot be distinguished from one another during life.

With the enlargement of the air-cells, and the destruction of many of the septa, a large number of capillaries have also perished, and the breathing-surface is materially diminished. The more points of contact the air finds with the blood, so much the more favorable are the conditions for interchange of gases. On the other hand, the smaller this surface is, so much the more incompletely, cæteris paribus, is the elimination of carbonic acid and imbibition of oxygen carried on. The loss of the alveolar septa, and the attenuation of the pulmonary tissue is, therefore, the first important factor in the dyspnœa from which emphysematous persons suffer. A sufficient renovation of the air contained in the air-vesicles is as essential to the normal performance of the oxygenation as is a due extent of respiratory surface. If the thorax and lungs do not expand properly during inspiration, or if they contract imperfectly during expiration, the air within the air-cells is not sufficiently renewed, and neither can the carbonic acid formed within the body be eliminated from the blood, nor can the latter obtain the oxygen required by the system.

That respiratory expansion and contraction of the lungs are embarrassed in emphysematous persons, has been maintained by most authors, and they, moreover, have connected the fact with the loss of elasticity of the pulmonary tissue; but, on the one hand, they have not sufficiently understood that the loss of the elasticity of the lung is due to certain changes in its structure, that it is dependent simply upon attenuation of the parenchyma, and loss of the elastic elements of the tissue; on the other hand, they have circulated many inaccurate and false notions as to the mode in which the respiratory motions are affected by decreased elasticity of the lung, so that it behooves us to study this influence more closely. It need hardly be mentioned. that the inspiratory enlargement of the thorax, by which alone expansion of the lung is effected, cannot possibly be impeded by the diminished elasticity of the lungs. The inspiratory muscles, among other obstacles, have to overcome this pulmonary elasticity. Hence, should this be diminished, inspiration cannot be embarrassed thereby; on the contrary, it should now proceed with greater ease than under normal conditions. It is much harder to decide the question whether, how, and how much decreased resiliency of the lung can interfere with expiratory contraction of the thorax. In the first place, when we look at the thorax proper, it seems unlikely that the ribs and the sternum need the suction of the elastic lung in order to return from their inspiratory to their expiratory state. The weight of the chest-walls and the spring of the ribs, which is overcome during inspiration by muscular action, seem to be quite enough to neutralize the inspiratory expansion of the thorax, when this muscular action is suspended. Experiments, which I have caused to be made upon the bodies of adults, have at least shown that, after perforation of the intercostal spaces, and after entrance of air into the pleural cavity, the thorax does not enlarge.*

There could not fail to be such an expansion, if the traction of the elastic lung contributed to the establishment of the position of expiration; since, in these procedures, the suction, which it produces upon the inner wall of the thorax, is suspended upon the entrance of air into the pleural cavity, and the collapse of the lung. Hence, as, in spite of emphysema, and in spite of the decreased elasticity of the lung, the thorax is still able to return to its expiratory state, as it needs no aid from the lungs to accomplish this, an emphysematous lung cannot afford any resistance to the contraction of the chest. It is very remarkable that we see mention made, in most pathologies, of a pressure made by emphysematous lungs against their neighboring parts, particularly of a displacement of the heart by the lungs, or of depression of the diaphragm and liver. If an emphysematous lung did indeed exercise a pressure inward and downward, did it really dislocate the heart and the liver, it is to be presumed that this pressure would also act outward upon the inner wall of the chest, and oppose an obstacle to its contraction during expiration.

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* In children this may be otherwise. From the greater flexibility of their thorax, it is not improbable that it is drawn inward during expiration by the draught of the elastic lung, and is contracted more by this means than by the spring of the ribs.

The supposition of centrifugal pressure of an emphysematous lung against its neighboring parts is due, partly to erroneous physiological, pathological, and anatomical assumptions, and in part to confusion of emphysema with that condition which we have described as permanent inspiratory expansion of the air-cells, and upon whose distinctness from emphysema we have already insisted.*

This demonstration will serve to show that emphysema has no influence in impeding either the inspiratory expansion, or the expiratory contraction of the *thorax proper*. The respiratory movements of the diaphragm, however, are very differently affected, in this respect, from the movements of the ribs. The diaphragm, which descends during inspiration upon relaxation of its muscles, returns to its inspiratory position, partly through the upward pressure of the abdominal viscera, partially by the traction of the elastic lung upon its upper surface; but it can easily be proved that the latter force plays the most important part in this process. In those cases of very relaxed abdominal walls occurring after repeated pregnancy, where all pressure upon the diaphragm on the part of the abdominal viscera is out of the question, the diaphragm still ascends during expiration, nay, as long as the thorax remains closed, it preserves this position in the cadaver, even after all the viscera of the belly have been removed. It does not relax and sink until the thorax is opened, because the suction of the lung, upon its upper surface, does not cease until then. Hence it is clear that the loss of elasticity, which the lung suffers by emphysematous attenuation of its tissues, may considerably impede the return of the diaphragm to its expiratory state, and sometimes may completely prevent it. Thus the coöperation is lost of the most important muscle of respiration, upon the free exercise of which the expansion and contraction of the chest, and with it the renovation of air in the vesicles, mainly depend; and we may designate, as the *second* great factor of the dyspnœa of emphysematous persons, the *permanently inflated condition*, or, as more commonly is said, the *permanently depressed state of the diaphragm*.

To these, however, in many cases, if not in all, a third cause is to be added. In many emphysematous patients the structural alteration of the lung is complicated with deformity of the costal cartilages, to which *Freund* first called attention in his valuable work. This deformity consists in a hypertrophy, by which each cartilage gains in volume in all directions simultaneously, assuming a remarkably firm, brittle, rigid character. By elongation of the costal cartilages, not only is the sternum driven farther from the ends of the bony ribs and pushed forward, but the ribs are moved upward and outward, and twisted upon their long axes in the same manner as they are turned about their axes during inspiration by the traction of the inspiratory muscles. As, however, the hypertrophied costal cartilages have become rigid, the thorax cannot return to its expiratory state, from this inspiratory ectasis, which may much exceed the largest degree of inspiratory expansion which the thorax can ever normally attain. A condition arises which *Freund* very aptly calls "rigid dilatation of the thorax." I believe that *Freund* goes too far in assuming the alterations of the costal cartilage to be the primary disease, and the structural change of the lung to be secondary. In a very few cases at most, principally cases of inherited emphysema, does this sort of genetic connection seem somewhat probable.

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* In the permanent inspiratory expansion of the air-cells, resulting from obstruction of the more minute bronchi, the air, which is confined in the cells, and compressed during expiration, does indeed hinder the thorax from returning to its normal condition of expiration.

In the majority of instances, as before said, it would seem to be only a complication. True, it is not an accidental one. It would rather appear that the same evils which effect the structural changes in the lungs also cause hypertrophy and degeneration of the costal cartilages. From its analogy to hypertrophy, and to alteration of other bodies, particularly to degeneration of the arterial walls, which is of inflammatory origin, or at least depends upon processes related to inflammation, we may suppose that this alteration of the costal cartilages is a result of repeated injury from distention and straining. When emphysema arises from forced inspiration, or from forced expiration, with closure of the glottis, from violent coughing, or through playing upon wind-instruments, the ribs also are expanded and strained, and the structural changes mentioned above develop in them in consequence of this irritation. If forced inspiration have produced the emphysema, the alteration of the costal cartilage is general. When forced expiration with constriction of the glottis is its cause, the alteration is confined to the upper ribs. Finally, costal hypertrophy and rigid dilatation of the chest, as a rule, do not take place at all where the emphysema does not appear until late in life, after the cartilages have become ossified. The importance of the part played by this rigid thoracic dilatation in many cases of emphysema is shown, among other ways, in our not unfrequently observing patients, whose dyspnœa decreases when they lie upon their bellies, and thereby compress the thorax; and others whose sufferings are greatly relieved by exerting a lateral pressure upon the lower part of the chest. In such patients I have frequently been able to observe, during life, that, in order the better to compress the thorax during expiration, they contracted the triangularis sterni, and the upper part of the transversus abdominis, and the expiratory gullies in the liver, first accurately described by *Liebermeister*, could be seen upon autopsy.. When rigid dilatation of the thorax is associated with structural change of the lung, it constitutes a third factor in the dyspnœa of emphysematous persons.

The entire aspect of the patient betrays obstruction of the respiration, oppression, dyspnœa, and want of air. They summon all their force to open out their thorax; the alæ nasi play; the lower part of the neck becomes harder and broader from the energetic contraction of the scalini with every inspiration. Frequently, and chiefly in the instances in which the costal cartilages are ossified and their joints atrophied, the forms of the sterno-cleidomastoidei stand out like hard cords. The muscular relaxation, the flabbiness, the apathy, which we perceive in all patients of this class, are due to imperfect decarbonization and retarded oxygenation of the blood. When to these constant hinderances to respiration a fourth one is added, as when the bronchi are contracted by an aggravated catarrh, the dyspnœa rises to an extreme pitch. Patients pass entire nights in their arm-chairs, fearing to choke if they should lie down. The complexion becomes ashy and muddy, the expression of the eye fatigued, the sensorium benumbed, the pulse and heart-beat small and irregular, the extremities cool; the manifestations of surcharge of the blood with carbonic acid increase to those of acute poisoning by this pernicious gas. Before the days of *Laennec*, such attacks, which recur with greater or less violence and frequence in all emphysematous persons, used generally to be considered and described as nervous asthma.

The derangements of circulation which are produced by the anatomical changes in the pulmonary parenchyma of emphysematous patients present a second series of symptoms. With the disappearance of the interalveolar septa and the loss of the numerous capillaries, the number of efferent channels from the right side of the heart is diminished. It follows hence, in the first place, that in that portion of the lung spared by the emphysema—usually its lower part—the pressure of blood is increased, and it becomes the seat of intense hyperæmia, and that chronic catarrh takes place in its bronchi, and often chronic œdema in its alveoli. Now, we might suppose, the number of capillaries which remain not being sufficient to receive the contents of the right ventricle, that the right ventricle, its auricle, and the veins of the aortic circulation would be overloaded with blood, and that cyanosis and dropsy would accompany very extensive emphysema. No indication, however, of any of these symptoms is usually observed for a long time, and if, from time to time, upon aggravation of the bronchial catarrh, œdema and cyanosis set in, the symptoms subside as the catarrh improves, a proof that the circulatory disturbance depends upon the transitory aggravation of the catarrh. The same causes operate in retarding derangement of the aortic system in emphysema, which often so long avert the appearance of cyanosis and dropsy in valvular disease of the mitral. In proportion as the circulation becomes embarrassed, a complication develops in the right side of the heart, which has the opposite effect, which counterpoises and compensates for the circulatory impediment—namely, *hypertrophy of the right ventricle of the heart.* The addition of symptoms of venous derangement about the vena cavæ does not take place in emphysema until this compensation begins to fail through a gradual degeneration of the thickened heart-wall by fatty metamorphosis of its muscular fibre. Then the jugular veins swell up and throb, with every ventricular impulse, as the vibration, into which the valves of the tricuspid are thrown during the systole of the right ventricle, is shared by the column of blood which rests upon it. (See chap., mitral insufficience.) The face becomes cyanotic, the lips swell up and become blue, the cheeks and alæ nasi have a varicose appearance. The obstructed evacuation of the cerebral veins makes the patient complain of dizziness and headache. All the signs attain their highest pitch when the patient coughs. Symptoms of engorgement in the course of the ascending vena cava also set in. The liver swells because its outflow of blood is impeded, and the engorgement extends through the portal system to the gastric and intestinal veins, giving rise to gastric and intestinal catarrh. In the same manner, the veins of the rectum often enlarge into varices (blind piles). The latter circumstance is almost always greeted with joy by the patient. They believe that they have now found the centre, the main root of all their trouble, and are all-hopeful for a cure from the critical hæmorrhoidal flow, just as they are misled by the gastric catarrh, and the loss of appetite which attends it, to regard the stomach as the source of their troubles, and to call their cough a "stomach-cough."

In the latter stages of emphysema the cyanosis often becomes extremely intense. The cheeks, ears, lips, and tongue of the patient are literally blue. In no other disease does the cyanosis attain such severity, excepting in cases of disorder of the orifices of the right heart, which are mostly congenital, and it is never met with in valvular disease of the left ventricle. True, in the latter, the patients generally grow blue about the lips and cheeks, but the general color of the countenance remains pale, and the blueness is never so pronounced as in emphysematous persons, or in such as have congenital heart-disease of the right side. This fact, which hitherto has received too little attention, is easy of explanation. In valvular affections of the left heart the pulmonary circulation is surcharged with blood, while the quantity of blood in the aortic system is abnormally small. On the other hand, in emphysema where many of the pulmonary capillaries have perished, and in congenital malformation of the right heart where the ventricles are usually ill developed, or have their orifices contracted, *it is the greater circulation which is overloaded, and the smaller which contains too little blood.*

This obstructive engorgement of the great veins extends also to the thoracic duct. When the subclavian vein is filled to distention, the flow of lymph and chyle must encounter a resistance equal to that opposed to the current of any other vessel which empties into the subclavian. Nay, if lymph be the source of the fibrin in the blood, we see, upon simply physical grounds, why the blood of emphysematous patients is poor in fibrin, why the "venous crasis prevents hyperinosis and increase of fibrin." Restricted afflux of chyle must, moreover, prejudice nutrition both of the blood and of the entire organism. It is one of several causes which contribute to the general emaciation and to the premature marasmus of emphysematous persons; perhaps, too, it may account for the lack of albumen in the serum of the blood, which produces a tendency to the establishment of dropsical symptoms.

As soon as the circulatory derangement ceases to be properly compensated for, symptoms of insufficient afflux of blood into the left side of the heart add to those of venous engorgement. Incomplete filling of the left heart produces a small pulse, a pallid complexion, and finally distinct diminution in the urine, as the amount of the urine depends chiefly upon the filling of the renal arteries and glomeruli of the malpighian capsules. The scantily secreted urine is concentrated, thick, and dark; the urates, which require a great deal of water for their solution, precipitate with readiness, in the form of a brickdust-like sediment. Precipitation of the urates is not due to concentration of the urine alone, nor to a relative increase in the quantity of the salts, but it may also depend upon their absolute increase, upon the formation of uric acid at the expense of the urea, the scanty supply of oxygen being insufficient to oxydize the nitrogenous products of the transmutation of tissue so as to produce urea, but only to such a lower degree as to give uric acid.

All other symptoms attributed to emphysema belong to its complications. The cough is a symptom of the chronic bronchitis, and often disappears altogether during the summer, while the emphysema continues as usual. In by no means all cases does physical examination give any fixed basis for the recognition of emphysema, and, when of small extent, its existence cannot ever be physically proved.

Inspection, with regard to the formation of the thorax, may give a negative result even in very intense emphysema. Individuals with a loose, long, so-called paralytic thorax often enough suffer greatly from the disease. This will not seem strange after our having shown that emphysema has no effect upon the expansion or contraction of the thorax proper. In some cases, indeed, the thorax shows characteristic abnormities, upon which we have founded the idea of a peculiar form of the chest, "the emphysematous thorax." In this the circumference and the depth, particularly in its upper and middle portion, are considerably increased.

Instead of presenting a broken line, the sternum forms a segment of a circle; the upper ribs are abnormally crooked, and bulged outward; the shape of the thorax is more spherical or barrel-shaped. It is remarkable that absolutely erroneous views as to the mode of origin of the emphysematous thorax have prevailed, while the real manner in which it is produced is as clear and simple as possible. The deformity of the chest is not produced by the emphysema of the lung, for both emphysema and deformity arise from the same causes. The emphysematous thorax is only seen in that species of the disease which arises from forced expiration with constricted glottis, as when playing upon wind-instruments, coughing, etc. By these acts the diaphragm is forcibly driven up by pressure of the abdominal muscles upon the viscera of the belly, and the air contained in the lung is strongly compressed. According to simple physical laws, as long as the costal cartilages retain their pliability, the thorax, like any other cavity with yielding walls, must become rounded and approximate to the spherical form, wherever the pressure upon it from within is augmented. But as the lower part of the thorax, to which the abdominal muscles are attached, is fixed, it cannot join in the rounding out of the middle and upper portions, and this explains why the emphysematous thorax is more barrel-shaped than spheroidal. By very deep inspirations, indeed, we can temporarily expand the chest to a considerable extent, but the thorax of a healthy man in full inspiration is differently shaped from a so-called emphysematous thorax. We may, for a short time, produce the latter in ourselves by making the strongest possible expiratory effort, while at the same time holding the nose and mouth. The sole reason for the permanence of this condition in emphysematous persons is the hypertrophy and alteration of the costal cartilages mentioned above. But this deformity of the chest, called *par excellence* emphysematous thorax, is not the only one observed in emphysema. In the cases in which emphysema develops in consequence of long-continued forced inspiration, we do not find this rounding of the upper and middle portions of the chest, in which the lower takes no part; but we find the lower portion dilated, and in a permanent inspiratory condition. The augmented contractility of the hypertrophied muscles of inspiration may contribute in some degree to this perpetual condition of dilatation, but it no doubt mainly depends upon the disease of the costal cartilages so often referred to.

Inspection, in many cases of emphysema, further reveals that, with every violent cough, a tumor projects through the upper aperture of the thorax at the side of the neck, which disappears immediately upon the cessation of the coughing-fit. I have recently satisfied myself that this tumor very rarely consists of the apex of the lung, which, with the pleura, has been driven into the space between the first rib and the neck by the violent pressure of the diaphragm, which, as it were, makes the chest too small. In the great majority of cases, these tumors are formed by enormous enlargements of the sinus of the jugular veins which fill up during the cough and empty themselves again when it ceases.

Finally, upon inspection, and, still better, upon palpation, we can perceive a strong concussion of the epigastrium, which is synchronous with the pulse, and which extends to the lower part of the sternum and of the adjoining ribs. This concussion has been usually ascribed to the shock of the apex of the heart, displaced toward the middle line of the body, but I agree with *Bamberger*, that such displacement is neither proved by facts nor even physically possible. This concussion of the epigastrium is not directly dependent upon emphysema, but is due to hypertrophy of the right heart, which complicates that disease, and it is found in all considerable cases of hypertrophy, especially of the right heart without emphysema. (See Hypertrophy of Heart.) Even when the heart of an emphysematous patient has become considerably enlarged, the shock of its apex is imperceptible, the lung having become interposed between it and the thoracic wall. Sometimes, however, besides the concussion of the epigastrium, a feeble heart-shock is perceptible at a point farther downward and outward than is normal. That the apex is displaced downward, is simply owing to the depression of the diaphragm, upon which it rests. But it is not only the apex of the heart, but its base too, which rests upon the diaphragm, and, indeed, upon the very part of it which descends the furthest in cases of its abnormal depression (farther, at least, than the point upon which the apex lies). Hence, as a natural consequence of depression of the diaphragm in emphysema, the oblique position of the heart becomes more horizontal, and its apex lies farther out.

Percussion affords an almost certain basis for diagnosis where emphysema is of considerable extent. However, we must not expect the sound to be unusually loud or full in all cases, as, if the resistance of the thoracic wall be augmented, even though the vital capacity of the lungs be increased, no very active vibrations, capable of producing any very loud or full resonance, can take place. Neither does the sound upon percussion become tympanitic, unless there be complications through which the pulmonary tissue entirely loses its elasticity. The tympanitic ring is a result of regular vibrations. If we make percussion upon a bladder, the walls of which are tightly stretched by inflation, the sound is not tympanitic, for the compression of the air, which is augmented or diminished every moment by the vibrations of the walls, prevents the occurrence of regular vibrations. It is precisely the same in a lung, which has become but a cluster of inflated cysts. The tension of the alveolar walls generally remains sufficient, even in very intense emphysematous disease of the lungs, to prevent the occurrence of regular vibrations. The only characteristic symptom of the disease, furnished by percussion, is abnormal extent of the full, clear sound of the lungs, as this proves that the diaphragm is depressed, which, as we have seen, is a necessary consequence of emphysema. While under normal conditions, the percussion-ring, corresponding to the lower limit of the right lung, reaches to the sixth rib at the right mammillary line, and passes over at this point into the dull percussive-sound of the liver, which here lies against the wall of the chest. In severe emphysema, the right lung pushes back the border of the liver considerably; and we sometimes hear clear percussion-sound almost as far down as the lower edge of the arch of the ribs. Upon the left, the dulness produced upon percussion over the heart commences normally at the level of the fourth costal cartilage. In well-marked emphysema of the left lung it spreads downward, often to the sixth costal cartilage, and in the most extreme cases the heart is so completely covered by the lung that the cardiac dulness has disappeared.

In auscultation, we must discriminate between the phenomena proper to the emphysema and those which are to be placed to the account of the accompanying bronchial catarrh. As a rule, it is said that the vesicular breathing is feeble or inaudible, in striking contrast to the intense percussive resonance. This assertion, however, is only so far correct in that, as a rule, besides the emphysema, there is usually a catarrh of the minuter bronchi. At the points where these complications coexist, we usually, indeed, hear nothing but rhonchi and fine moist *râles*, or, at the utmost, very feeble vesicular breathing. At points, however, where there is no catarrh, where the air passes freely from the bronchi into the dilated cells, the respiratory murmur is generally remarkably loud and hissing. It is very commonly found that we can only hear rhonchi and *râles* in the lower lobes of the lungs; and in a manner strikingly in contrast with this, particularly at the anterior wall of the chest in the neighborhood of the sternum, the respiration is loud and hissing.

This circumstance, which we had attributed to the collateral œdema of the healthy portion of the lung, which is usual in emphysema, is ascribed by *Seitz*, of Giessen, in some degree, to the descent of the secretion by gravitation from the upper to the lower regions of the lung (a theory which has much to recommend it). The murmurs sometimes audible in the heart, even where there is no valvular disease, are to be considered in treating of degeneration of the heart; but I may say that I fully corroborate the observation of *Seitz*, that the heart-sounds, though remarkably feeble at the level of the third and fourth ribs, where the organ is covered by the lungs, are very audible at the epigastrium. The explanation is manifest.

The disease may commence in childhood, and continue throughout life. Many emphysematous persons reach even an advanced age, although their troubles grow with their years, the dyspnœa augmenting, the asthmatic attacks increasing in violence and frequency. The sufferers are never cured. If they feel better in summer, this is because of the remission of the accompanying catarrh, and the decrease of the dyspnœa, as far as it depends upon these complications. The part which chronic catarrh plays, not only in the dyspnœa, but in the cyanosis and dropsy of the emphysematous, is, as we have repeatedly explained, a very considerable one. Death finally takes place (if the patients do not meanwhile succumb to an intercurrent malady), with the symptoms of marasmus or of general dropsy. The patients very rarely die of an asthmatic attack.

DIAGNOSIS.—Emphysema of small extent cannot be diagnosticated with certainty. Cases of considerable severity, which lead to violent dyspnœa and cyanosis, are easily to be distinguished, by physical examination, from other conditions which give rise to those symptoms. Of the distinction between pneumo-thorax and emphysema we shall speak hereafter.

For the differential diagnosis between vicarious and substantive emphysema, the history of the case and the physical signs—in some cases at least—furnish ground. If the emphysema have developed after a pneumonia or pleurisy, if no particularly violent cough have preceded it, or if the patients distinctly affirm that the shortness of breath is of earlier date than the cough, we may infer, with a certain degree of confidence, that a partial wasting of the lung or adhesion of the pleura has occasioned a vicarious emphysema in the anterior and lower part of the lung, spared by the atrophy, or that the disease depends upon a primary structural wasting. On the other hand, should we find emphysema in a person who has been a musician or postilion, who boasts of having played well, and of having been able to keep up the note for a long while, or if the shortness of breath have arisen after whooping-cough, or a tedious catarrh accompanied by violent cough, the presumption is warranted that we here have to deal with a substantive emphysema. In like manner, a permanent inspiratory state of the thorax argues for the former, the barrel-shaped chest more for the latter form of emphysema.

PROGNOSIS.—The prognosis as to life is, upon the whole, favorable. A fatal termination of the disease is rare, and then only occurs after long duration. Indeed, it cannot be denied that emphysema sets up a certain degree of protection, if not absolute immunity against tuberculosis, depending either upon the "venous crasis," or upon the bloodlessness of the lung, especially at its apex. Emphysema complicated by pneumonia (a somewhat unusual occurrence) should excite our solicitude, lest the pneumonic exudation be not reabsorbed, but should descicate, and afterward break down, together with the cell-wall, after undergoing cheesy metamorphosis. (See Croupous Pneumonia.) Prognosis as to complete recovery is altogether unfavorable, as shown above.

TREATMENT.—The causal indication requires judicious treatment of the bronchial catarrh, whooping-cough, etc., so as to set bounds, at least, to the progress of the emphysema, though a cure be impossible. In raw weather, and in winter, when the temperature is low, make emphysematous persons keep constantly in their chamber. Observant patients are sometimes able to tell precisely what degree of cold is hurtful to them, and forbids their going out. To meet the indicatio morbi, the periodical administration of emetics has been advised. The object was to compress the distended vesicles through the pressure exerted upon the lung by active vomiting and retching, in the hope that frequent repetition of the process might effect gradual decrease in their size. By others, tonics are recommended, to brace the relaxed pulmonary tissue and make the alveoli smaller. These, and many other proposed modes of medication equally naive, and quite as false in principle, do not deserve the least confidence as radical "cures" of emphysema. The nutritive alterations upon which the disease depends are irreparable, and we are totally unable to fulfil the indications for the disease itself.

The symptomatic indication, first of all, requires proper treatment of the bronchial catarrh, which almost always accompanies emphysema, and greatly adds to the distress of the patient. Habitual wearing of flannel next the skin, stimulants to the chest, warm baths of water or vapor, the alkaline muriatic mineral springs, especially the thermal springs of Ems, and other similar treatment, are often of signal benefit to emphysematous patients in relieving their sufferings for a while, but their action is all due to their timely application against the most serious complication of the disease, the dry catarrh.

The next symptomatic indication is to moderate the habitual shortness of breath of the patient and the attacks of severe dyspnœa, for brevity's sake, called asthma. In order to allay the persistent oppression of the chest, it is very desirable to send the patient for the summer to the pinewood region, and particularly to places where there is a heavy fall of dew. The benefit which they derive in this highly-oxygenated atmosphere is always warmly extolled by them. From experiments made with apparatus for inhalation of compressed air, the effect of this unfortunately somewhat costly remedy, both upon the avidity for air and the general health of emphysematous patients, is excellent, although only palliative. Many of the patients "feel like new men" while in the machine. The explanation of the improvement is easy. We have already stated our views of the main element in the benefit derived from inspiration of compressed air.

To avert the asthmatic attacks, the patients must observe a strict diet, avoid food likely to induce flatulence, eat little before going to bed, and keep the bowels open daily. For the latter purpose, the pulvis liquoritiæ compos. is a mild and efficient cathartic. During the attack, beware of mistaking the blunted sensibility and other "head-symptoms" for the effects of venous engorgement of the brain, and thus bleeding the patient. The symptoms of carbonic-acid poisoning would only be promoted by depletion. The narcotics, too, especially opium, must be used with caution in these attacks of emphysematous asthma, unless called for by bronchial spasm. The more suitable remedies (besides the emetics, which are very appropriate) are the stimulants, camphor, musk, benzoin, and the large doses of port wine proposed by *Waters* (℥ j—℥ iss every three hours), and when these fail, the use of turpentine (ʒ i to ℥ ss every three hours) in an aromatic water.

For the dropsy, as has already been stated, whenever it depends upon a capillary bronchitis, I have repeatedly produced excellent results by means of vigorous diaphoresis. Later in the disease, when dropsy arises from failure of the heart to compensate for the circulatory derangement of the lungs, it may be relieved for a time by the use of digitalis (an infusion ℈ss—℈j to water ℥ vj), just as in dropsy from valvular disease of the heart. Where digitalis fails, squills sometimes does excellent, though merely transitory, service. (Acet. sillæ. ℥ j; pot. carb. q. s. ad. saturationem. Aquæ destillat. ℥ vj. ♏. S. a tablespoonful every two hours.)

## CHAPTER IV.

### DIMINISHED CAPACITY OF THE AIR-CELLS—APNEUMATOSIS—ATELECTASIS—COLLAPSE—COMPRESSION OF THE LUNGS.

Etiology.—There are conditions under which the capacity of the air-vesicles decreases and their walls finally come into contact. This latter state, which is normal during fœtal life, may persist in portions of the lung after birth. It is then called congenital atelectasis. In other cases the air is absorbed, at a later period, from a number of the vesicles, which then collapse. This is called acquired atelectasis, or collapse of the lung. In other instances the air is expelled from the vesicles by external pressure. We then speak of compression of the lung. Congenital atelectasis is most frequently found in feeble children, particularly those who have been born prematurely, or who have come into the world in a state of apparent death after tedious labor. It would seem as though air-vesicles, which do not become filled with air immediately after birth, are subsequently more difficult of inflation, so that children, which are not induced to cry, and thereby caused to make deep inspirations during the first hours of their life, very commonly suffer from atelectasis. In other instances a catarrh, either congenital or contracted in the first hours of life, seems to have given rise to atelectasis by contracting or occluding some of the bronchi, and thus impeding the supply of air to the vesicles to which they lead. Collapse of the lung, or acquired atelectasis, is always connected with acute or chronic bronchial catarrh, and is of somewhat frequent occurrence in children, as their bronchi are small and easily obstructed. In adults it is a peculiarly frequent complication of the catarrh, which is one of the symptoms of typhus (typhoid?). Compression of the lung takes place in consequence of the presence of liquid or air, more rarely of a tumor in the pleural sac, from effusions in the pericardium, from aneurisms, curvature of the spine, arrested development of the thorax, and finally, from voluminous effusions in the abdominal cavity, by which the diaphragm is driven upward.

Anatomical Appearances.—In congenital atelectasis circumscribed spots in the parenchyma, more rarely the half or the whole lobe of a lung, are found to be depressed somewhat below the level of the surrounding parts. These spots are of a dark-blue color, firm, do not crackle, and, when cut into, present to view a smooth surface abounding in blood. At first they may be readily inflated, afterward they become more rigid, contain less blood, and it is no longer always possible to inflate them. The areolar walls seem to be fast glued or grown together. The alterations of the parenchyma in acquired atelectasis are essentially the same as those just described. *Rokitansky* formerly called it catarrhal pneumonia. The blue, depressed spots here contrast more against the surrounding emphysematous lung. If we cut into the atelectatic spot, we usually come upon a thick, muco-purulent plug, which has stopped up the bronchus leading to it. When the atelectasis is of long standing, other changes take place in the collapsed portion of the lung, which belong to that very frequent sequel of this disease, catarrhal pneumonia, upon which we shall enlarge more fully in Chapter X. Compression of the lung of slighter grade presents an increased density and consistence, and a condensed condition of the parenchyma, which, however, is not entirely void of air. In the more intense forms of compression the air has left the vesicles and bronchi, but the pressure has not sufficed to overcome the blood-vessels and expel the blood. The condensed lung is red, full of blood, moist, resembling a piece of muscle, hence we call it carnified. In the highest grade of all, the vessels also are compressed, the lung appears bloodless, dry, gray, leaden, often converted into a gray, leathery, tough mass.

SYMPTOMS AND COURSE.—The symptoms of congenital atelectasis are essentially those of insufficient breathing and incomplete decarbonization of the blood, and have often been described. The child breathes softly and very quickly, is remarkably drowsy, does not cry as it should, but merely whines and whimpers, and cannot suck freely. Finally it grows pale, cool, and even cold, the nose becomes peaked, the lips livid or lead-color, and it usually perishes in the first few days of life; more rarely, not until the third and fourth week, with the symptoms of general paralysis, and much less commonly with convulsions. It is but seldom that we can succeed in demonstrating by percussion that solidified lung lies in contact with the thoracic wall, as the points of atelectasis rarely are very large. When collapse of the lung accompanies capillary bronchitis of little children, it cannot, in many cases, be diagnosticated with certainty. We have seen, in treating of the latter disease, that children may exhibit all the symptoms of insufficient breathing and carbonic-acid poisoning from closure of many of the little bronchial tubes, and without collapse of the air-vesicles. If, then, these symptoms should appear in the course of a capillary bronchitis, we are only warranted in diagnosticating an acquired atelectasis, if we find deadened resonance over a wide extent of the chest. Generally speaking, the collapsed spots are not of sufficient magnitude to make the percussion-sound dull. It most frequently arises in measles, in consequence of very extensive collapse of both lower lobes of the lungs, and presents a symmetrical dulness on each side of the spinal column.

It is difficult to discriminate between the symptoms of compression and those of the diseases which produce compression. The symptoms of imperfect respiration, where the pulmonary vessels also are compressed, are accompanied by derangement of the circulation similar to that which we have described as attending emphysema; distention, dilatation, hypertrophy of the right heart, subsequent distention of the veins of the greater circulation, cyanosis, venous engorgement of the brain, liver, and kidneys. In like manner the left heart, which is supplied by the uncompressed capillaries alone, receives too little blood, the pulse becomes small, the complexion pale, the urine scanty. In severe compression of the lung, the sufferers usually die of dropsy. Compression of a large section of a lung has an important influence upon the distribution of the blood in the uncompressed parts. If all the blood from the right heart be directed to one lung alone, the pressure in that lung is considerably increased. The greatest danger often threatens from œdema of the uncompressed lung, which may even demand venesection, etc. Should the lower parts of the lung be compressed by effusions in the peritoneum, the hyperæmia thus arising of the upper lobes (collateral fluxion) may produce danger and call for tapping. In the same way deformed persons, when a part of their thorax is contracted and the lung in the narrowed spot compressed, suffer from hyperæmia, catarrh, and œdema of the uncompressed portion.

It is a curious fact that humpbacked persons can breathe without difficulty, show no irregularity of the circulation, and do not evince symptoms of dyspnœa or of cyanosis until the period of puberty. This fact is easily accounted for, if we reflect that bones deformed by rachitis are retarded in their growth, even after the original disease has become extinct. Where the thorax and the thoracic vertebræ have been the principal seat of the disease, the crookedness and deformity of these parts at the time of its establishment may not, perhaps, have caused any loss of room in the chest; but if the rest of the body grow in the normal manner, the development of the chest being retarded, a disproportion must arise between the space, which, though sufficient for the lungs of a child, is insufficient for those of an adult, and for the dimensions of the remainder of the body and of the mass of blood belonging to it. Deformed persons then acquire the appearance and complexion with which we have become familiar in the emphysematous. They seem very short of breath, and usually die early in consequence of this respiratory and circulatory derangement, although it is exceptional for them also to become tuberculous.

Treatment.—Take care that newly-born children be made to cry properly, and that their mouths be freed from mucus; and should there be any accumulation of mucus in the bronchi, give them an emetic of ipecac and oxymel of squills. Should the respiration still remain imperfect, then, placing the child from time to time in a warm bath, apply a cold douche to the breast with a clyster-pipe. Do not let such children sleep too long and continuously, force them even to cry periodically if they do not cry of their own accord, by brushing the soles of their feet, etc. If they do not take the breast, see that they receive nourishment from the spoon, the mother's milk being the best. Finally, give a few drops of wine occasionally, and, if the child shows a tendency to become cool, do not let it sleep in a cradle, but rather in the arms of its nurse or mother. Caution and method are not uncommonly rewarded by surprising success.

The treatment of acquired atelectasis is the same as has been recommended for capillary bronchitis, when the latter disease has led to the obstruction of the finer bronchial twigs. In most cases, if we succeed in overcoming the obstruction, air will reënter the collapsed vesicles.

Compression of the lung requires mainly a judicious treatment of the principal disease, and symptomatic treatment of the more threatening derangement of the circulation.

## CHAPTER V.

### HYPERÆMIA OF THE LUNG—PULMONARY HYPOSTASIS—ŒDEMA OF THE LUNG.

ETIOLOGY.—Hyperæmia of the lung must be regarded as of two kinds—active and passive. For the first, *Virchow* has proposed the name of "fluxion"—"rush of blood"—(Wallung), while he calls the passive form stagnation of the blood—(Blutstockung). These names are the more desirable, since the words active and passive do not quite correspond to the physiological processes which give rise to the two forms of disease. *Fluxion*, indeed, depends more upon an increased, accelerated afflux; stagnation upon an impeded, retarded efflux from the capillaries, in whose contents we are especially interested, as it is upon the latter that both function and nutrition of organs depend.

I. *Fluxion, or determination of blood to the lungs*, is observed—

1. When the *action of the heart is increased.* We often see young persons at the period of puberty, particularly narrow-chested, overgrown subjects, in whom the most trifling causes, as the moderate use of stimulants, slight bodily efforts, and the like, produce palpitation of the heart, with considerable increase in the force of its impulse, accompanied by symptoms of pulmonary hyperæmia. But even without cardiac erythism, and where there is no special predisposition, over-violent bodily efforts, immoderate use of spirits, great mental excitement, rage, etc., may give rise to dangerous hyperæmia of the lung, together with increased and accelerated action of the heart. There are cases, unfortunately, as scandalous as they are of frequent recurrence, in which delirious persons, or patients with delirium tremens, having been brutally strapped to their bed, and intrusted to a rude nurse, are found dead the next morning, with bloody foam upon their lips. Such a case reveals, upon autopsy, an intense hyperæmia of the lung and pulmonary œdema, as the sole cause of death. The symptoms are difficult of explanation. In most of the organs in the greater or systemic circulation,

mere augmented action of the heart does not produce hyperæmia. The fuller the arteries, and the more distended their walls, so much less full are the veins, and so much is the tension of their walls lessened. The efflux from the capillaries is accelerated in proportion as the afflux is increased; the circulation is quickened without any actual increase of the quantity of blood in any of the organs. Whether another condition exists in the lungs, or whether increased heart-action alone is thus capable of giving rise to hyperæmia, we do not attempt to say, as we are not sufficiently acquainted with the normal condition of the pulmonary circulation, and even the natural degree of pressure of the blood in the pulmonary artery when the chest is closed is unknown to us.

2. The instances of fluxion to the lung produced by direct irritation, by the transitory action of cold upon the pulmonary tissue, the inhalation of very hot air, or of air mingled with irritating matter, are easier of comprehension. Here the tissue in which the capillaries are imbedded seems to be relaxed, and to oppose less resistance to the relaxation of the capillary walls. The same causes, acting upon the skin, produce fluxion upon it in a manner quite similar. The skin reddens if it be exposed to cold for a short time, as if a hot poultice or a sinapism had been laid upon it. The chronic fluxions, too, which accompany the formation and softening of neoplastic growths and tubercles, especially tuberculosis of the lungs, occur in the same way, and may be traced to abnormal relaxation of the connective tissue.

3. A third form of fluxion, as yet far too little noticed, but which we have already alluded to in treating of emphysema and pulmonary compression, occurs in all cases of obstruction of the pulmonary circulation from stasis in its capillaries, or where the latter are compressed or destroyed, and, of course, it must develop in portions of the lung where the circulation is free from impediment. Such *collateral fluxion* is a physiological sequel to ligation of an arterial trunk, as is proved by the appreciable dilatation of the surrounding unobstructed vessels, and it is quite indispensable to the symptomatology of most diseases of the lung. In the most simple manner it explains symptoms, which otherwise would be unintelligible, and it accounts for the action of venesection in pneumonia, pleuritic effusion, etc.

4. Finally, we have already stated that a rarefaction of the air in the alveoli produces determination of blood to the lungs, just as a cupping-glass or the boot of *Junod* causes fluxion to the skin. The suspended or diminished pressure to which the capillaries of the air-cells of a child with occluded glottis are subjected, when it expands its chest, is, as we have seen, the main reason for the consecutive bronchial catarrh and pulmonary œdema in croup, and of the poor success of tracheotomy.

II. *Stagnation of the blood, passive hyperæmia*, from which we illogically separate the mechanical form, takes place in all cases in which the pulmonary veins are abnormally filled and their walls unduly stretched. Here the blood flows from the capillaries with difficulty, while the arteries continue to convey blood to them, even though scantily filled themselves, since even then their walls evince a greater degree of tension than the capillary walls. (Blood continues to flow from the arteries into the capillaries after the heart has ceased to contract.) Hence we see that stagnation results in a far greater dilatation of capillaries than fluxion does, as when there is much obstruction of the venous current in the capillaries, which have become, as it were, blind appendices to the arteries, the blood continues to enter them until the tension of the capillary walls is equal to that of the artery, or until the delicate membrane can no longer support such a pressure, and becomes ruptured. Stagnation, or engorgement of the pulmonary capillaries, occurs most typically—

1. From contraction of the left auriculo-ventricular opening and insufficience of the mitral valve. Both forms of disease of the heart are accompanied by the most intense hyperæmia of the lung. We know that the brown color of the indurated hypertrophied lung depends upon rupture of the dilated capillaries, the chief cause of which we have found to be valvular disease of the mitral. Whether the evacuation of the auricle be retarded, or whether the blood be regurgitated during the systole into the auricle, either process must impede the emptying of the pulmonary vein, and give rise to overcharge of the capillaries.

2. Enfeebled action of the heart results in imperfect evacuation of its cavities, and hence in impeded efflux of blood from the veins. Here the supply from the arteries is not diminished in proportion as the outflow from the capillaries is obstructed, and thus asthenic fevers, in which the contractions of the heart are frequent, but incomplete, such as typhus, puerperal fever, or pyæmia, are constantly accompanied by engorgement of the pulmonary capillaries. When the heart's action is weakened, gravity furnishes a new impediment to the evacuation of the capillaries in dependent portions of the body. While such an obstacle is easily overcome when the heart contracts with energy, yet when its action is depressed, we soon see evidences of the effect of gravitation, and hyperæmia begins to form at the more dependent places. A healthy person may lie in bed for months without the development of this form of hyperæmia (hypostasis) in the capillaries of the back, or the formation of bed-sores, or the different phases of pulmonary hypostasis which are the almost constant accompaniment of a typhus of long duration.

We have learned that swelling and succulence of the mucous membrane, and increase and alteration of the follicular secretion, are the constant result of hyperæmia of a mucous membrane; similar processes take place in the alveoli in all cases of severe hyperæmia. Here, too, the walls swell up, become more moist and succulent, but the secretion, or, more properly, the transudation, which is poured into the cells differs from the bronchial secretion, being liquid and serous. If we bear in mind that there are but few mucous follicles even in the finer bronchi, and none at all in the air-cells, and that the structureless cell-wall is covered merely by imperfect pavement epithelium, it must be evident that the secretion from the vesicles, which have no mucous membrane proper, must be very different from bronchial mucus. While, in other organs, the term œdema is applied to an effusion of serum into the interstitial tissues, the term *œdema of the lung* is only used in cases where such infiltration is combined with an effusion upon the free surface of the lung, *i. e.*, into the pulmonary vesicles.

Œdema of the lung, however, is not, in all cases, a consequence of hyperæmia, or of increased pressure of the contents of the capillaries upon their walls, but, as in other organs, serum filters out of the pulmonary capillaries into the tissue and into the vesicles, under slight pressure, whenever the serum of the blood has but very little albumen in solution, or whenever a dropsical crasis has developed. We shall consider this subject more fully in treating of Bright's disease.

If œdema arise from a hypostatic hyperæmia, it is called hypostatic œdema. As we have learned, however, there is a double reason for the vascular engorgement in hypostatic hyperæmia, and hence it is easy to understand that in this form the capillaries become extremely overfilled, and that their walls undergo an excessive pressure. In this form of hyperæmia it is not merely a transudation of a solution of dilute albumen which takes place, but all portions of the serum of the blood, even the fibrin, pass through the now porous wall of the vessels, and we call this condition hypostatic pneumonia, a process which takes place simply from stagnation of the blood, and has nothing in common with inflammation proper.

Anatomical Appearances.—When the hyperæmia is moderate, the lung is bloated, dark red in color, and its vessels are filled to bursting. The tissue is succulent, relaxed, crackles but little, blood flowing freely over the cut surface; a bloody, foamy liquid is contained in the bronchi. When of longer duration and greater intensity, the parenchyma looks dark, bluish red or blackish red. The interstitial tissue and the alveolar walls are so much swollen that the condensed parenchyma scarcely gives any indication of its cellular structure. The lung, thus solidified, presents a certain similarity of appearance to the tissue of the spleen, and is therefore said to be splenified.

If œdema have developed in the lung, it seems swollen, does not collapse when we open the chest, and is tense to the touch. If recent, it does not pit on pressure; after longer duration, the parenchyma has lost its elasticity, and the lung retains the impression of the finger longer and more distinctly. If the œdema be consequent upon an intense hyperæmia, the œdematous lung is colored red, but, if it be one of the symptoms of a general dropsy, it may appear quite pale. If we cut into the œdematous spots, an enormous quantity of liquid, sometimes clear, at others of a red color, mixed more or less with blood, flows over the surface of the cut. This liquid is full of bubbles, frothy, and copiously mixed with air, if the air-cells have not been entirely filled up with serum and still contain air. In other cases, the liquid hardly contains any bubbles, except a few from the larger bronchi. Here the serum has expelled all the air from the vesicles. In cases of hypostasis we find the same conditions; intense hyperæmia, amounting to splenification, or a more or less complete œdema, uniformly occupying the posterior portion of the lungs next the vertebræ. If the patient have lain continually upon one or other side, the hypostasis is often confined to this side alone, and may be very extensive, while the other lung may be healthy. If the contents of the air-vesicles at the condensed portions of the parenchyma cannot be completely evacuated by pressure, if the section shows an indistinct granular aspect, if the liquid which flows out be clouded by little coagula of fibrin, we have the so-called hypostatic pneumonia before us.

SYMPTOMS AND COURSE.—A moderate degree of fluxion to the lungs presents no symptoms. The dilated capillaries present a greater surface to the air, the circulation is accelerated, and with this acceleration the change of the blood in the lung grows more brisk, as both circumstances promote and facilitate oxygenation. When, however, the fluxion is more considerable, the enlargement of the dense capillary net and the swelling of the cell-walls from augmented transudation may diminish the capacity of the air-vesicle. An obstacle to respiration is thus set up. The lungs cannot inhale so much air. Those narrow-chested youths and girls, of whom we have spoken, in their attacks of palpitation, complain of shortness of breath, nay, they very correctly call the sensation which they experience in the chest a "fulness" or "stricture." A short, dry cough is added to this condition; far more rarely a frothy expectoration, with scattered streaks of blood. There is no pain in the chest. Physical examination shows no abnormities. We may as well state here that "habitual determination of blood to the chest" is sometimes the forerunner of tuberculosis, though perhaps not as often as we are apt to believe.

The violent hyperæmia of the lungs, mentioned in the pathogeny, and which must be regarded as consequent upon excessive action of the heart, sometimes arises rapidly, and threatens life with unexpected suddenness. Hence such cases are called pulmonary apoplexy (*Lungenschlagfluss*). The shortness of breath quickly increases to a serious extent; the breathing grows hurried and scarcely to be counted. The feeling of fulness and compression causes fear of death and a sensation of choking; every cough fills the mouth with a copious, frothy, bloody expectoration. The heart beats visibly, the radial pulse and the carotids betray the tension of the arteries. The face is reddened. The œdema, which follows this form of hyperæmia, soon makes itself felt. The vesicles, filled with serum, can admit no more air; an acute surcharge of the blood with carbonic acid changes the scene. The restless patient becomes still and drowsy, the face paler, the muscles of the bronchi, palsied with the other muscles, cannot rid the tubes of their serous contents. Coarse, moist *râles*, audible even in the trachea, announce the approaching end, the threatening suffocative effusion.

The symptoms of acute fluxion, brought on by the inhalation of irritating gases, are modified by the coëxistence of irritation of the larynx and bronchial mucous membrane, and are accompanied by violent coughing-fits. The hyperæmia to which tuberculosis, cancer of the lung, etc., give rise, and which most generally produce pulmonary and bronchial hæmorrhage, are to be treated of in the next chapter.

Collateral fluxion to the lungs forms a grand feature in the description which we shall present of pneumonia, pleuritis, and pneumothorax. Here a large part of the dyspnœa depends upon the overfilling of the capillaries and swelling of the vesicles, in the portions of the lung unaffected by the inflammation. Without this complication, or, to speak more properly, if no such condition arose when the circulation is impeded, the unaffected vesicles could better obtain their supply of air. If the blood pressure be lessened by venesection, the collateral fluxion is reduced, the dyspnœa often completely disappears, although the chief disease continues unabated. When patients die in the first stages of pneumonia or pleuritis, or shortly after air has penetrated into the pleural sac, and compressed the lung, they die of collateral hyperæmia and collateral œdema. If we examine the records of post-mortem examinations, we shall not fail to find evidence of this form of hyperæmia, although it is but little appreciated in interpreting the symptoms.

Passive hyperæmia (*Blut-stauung*), even when unaccompanied by pulmonary œdema, creates greater dyspnœa than fluxion to the lung. Patients with insufficience and contraction of the mitral, even if they have no bronchial catarrh, and when the engorgement does not extend from the alveolar capillaries into their anastomoses so as to produce tumefaction of the mucous membrane and contraction of the tube, nevertheless usually suffer from a very distressing shortness of breath, aggravated by the slightest movement. This is easily accounted for, when we reflect that in passive congestion (*Blut-stauung*) the circulation is as much retarded as in fluxion it is accelerated, that in the former a double cause of dyspnœ aprevails, in the latter but one. Intense dyspnœa and all the symptoms of pulmonary apoplexy and suffocative effusion, which we have described, are often suddenly and unexpectedly added to the constant shortness of breath of disease of the heart. Effusion into the air-vesicles now exists beside swelling of their walls; the respiration, merely impeded hitherto, has now become inadequate. A great number of those who have disease of the heart die of acute passive congestion and acute œdema, without discoverable cause for the sudden increase in the impediment to the circulation. In other cases the symptoms of effusion of serum into the pulmonary vesicles, the inadequate respiration, and final death of the patient, take place more gradually in cases of disease of the heart.

If, in the course of an asthenic fever, whether it be a symptom of typhus or of pyæmia, the respiration should become shallow and incomplete, should percussion indicate a condensation of the parenchyma of the lung, near the spinal column, should sputa be ejected more or less tinged with blood, we have to do with an obstructive engorgement of the lung with hypostasis or with its sequelæ.

It would be unnatural and artificial to make a distinction between the symptoms of hyperæmia and of œdema. If a hyperæmia be intense, œdema occurs as one of its most important symptoms. We infer that this normal and necessary result has taken place, in the first place, from the grade of the dyspnœa, which never becomes so severe from swelling of the alveolar walls alone, as from œdema. Almost universally where hyperæmia has produced death, serum has been found in the air-vesicles.

The characteristic sputa give a second point for diagnosis. Such liquid secretion is seldom or never discharged from the bronchial mucous membrane, and the expectoration of liquid transparent, profuse, sputum, more or less mixed with blood, if it supplant the viscid, scanty sputum of pneumonia, is very properly regarded as of serious omen. Auscultation also gives information as to the occurrence of œdema. A dry *râle*, that is to say, a *râle* which is formed in viscid fluid, may easily be distinguished from a moist one, that is, from a rattle which takes place through the medium of a thin liquid. In the secretion of the bronchial mucous membrane we seldom hear such moist rattling sounds occur as those which arise when the serous transudation of the vesicles fills up the bronchi. In other cases we hear no respiratory sound in spots at which the vesicles are filled up by œdema, and where no air can enter. Bronchial breathing is only to be heard in rare instances.*

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* Bronchial breathing takes place when the vesicles, filled with serum, do not

Percussion, finally, which undergoes no change from hyperæmia alone, sometimes indicates that œdema has supervened upon hyperæmia. The sound upon percussion when the alveolar walls have lost their elasticity through œdema, and are but lightly stretched over their contents, is sometimes distinctly tympanitic. If, however, all the air have been driven out of the air-cells by the œdema, and the lung have become void of air, the sound upon percussion becomes dull and flat, as with every other condensation of the lung. If these manifestations appear in the characteristic places for hypostasis, we have to do with this form, or with its sequelæ.

With regard, finally, to that form of œdema of the lung which arises in general dropsy, the appearance of dropsical swelling of the subcutaneous cellular tissue and of effusions in the serous cavities furnishes the best grounds for interpretation of the dyspnœa which accompanies these symptoms. Should serous sputa, moist rattles, a tympanitic or dull sound, upon percussion, supervene, we are warranted in regarding pulmonary œdema as their cause.

DIAGNOSIS.—Hyperæmia and œdema of the lung, if we keep in view the symptoms just described, are easily distinguished from other diseases of the lung. It may, however, be very difficult (easy as the matter may appear in the study) to make a distinction at the bedside between active and passive hyperæmia, between fluxion and obstruction; and, moreover, confusion of the two may lead to the worst consequences, to mistakes which not unfrequently threaten the life of the patient. The confusion occurs most frequently between the collateral fluxion, occurring in the course of a pneumonia and pleurisy, and the passive hyperæmia, to which enfeebled heart-action and asthenic fever give rise. We so frequently notice the occurrence of passive pulmonary hyperæmia and œdema of the lung, upon the final exhaustion of the patient, upon the diminution of the pulse, upon the delirium, and the dry tongue, that we are apt also, in cases of recent pneumonia, if the pulse be small, and the patient delirious, to think of passive hyperæmia and of obstruction from commencing paralysis of the heart, and instead of venesection to prescribe wine, camphor, and musk.

In treating of croupous pneumonia we shall go more fully into detail upon the subject of pulmonary fluxion and engorgement, symptoms of the utmost importance in that disease, and demanding especial consideration in its treatment.

PROGNOSIS.—The prognosis of hyperæmia and of œdema of the

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contain any air, but where, at the same time, the bronchi, which lead to the condensed spot, are not filled up by the secretion. It is easy to see that the latter condition, requisite for bronchial respiration (of the origin of which we shall speak more fully while treating of pneumonia), is almost always wanting in pulmonary œdema.

lung depends essentially upon its exciting causes. Fluxions, if they do not proceed from adventitious productions in the lung, are generally of less serious character, and are more amenable to treatment than obstructions, the causes of which are usually difficult to allay. Prognosis of the various forms can be derived from the description of the course of the disease.

TREATMENT—*Indicatio causalis.*—As increased action of the heart is a frequent cause of fluxion to the lung, and as, in youthful subjects, habitual palpitation of the heart, accompanied by hyperæmia of the lung, is often the forerunner of tuberculosis, a regimen and treatment suitable to such condition are demanded. Strictly forbid the use of spirits, tea, coffee, and order all food or drink to be allowed to cool somewhat before it is taken. In like manner inexorably forbid dancing and riding, and other violent bodily exertion, at the same time enjoining regular and moderate exercise. Shield the patient, as far as possible, from all psychical excitement. Besides these precautionary measures, the lung is to be protected from injury. Let all hot and smoky rooms and all dusty places be avoided, and do not let the patient inhale very cold air. Acidulated drinks, lemonade, cream-of-tartar water, are to be recommended. The milk and whey treatment is especially suitable for such cases, and, above all, the "grape cure" of Dürkheim, Meran, on the lake of Geneva, and other places with a mild clime, where sweet grapes, which do not purge, are cultivated.* It merely hastens the end of patients, in an advanced stage of phthisis, to remove them from the quiet and comforts of home in order to try the grape or whey cure. On the other hand, these cures often do most brilliant service in the instances under discussion, which may not incorrectly be regarded as cases of incipient tuberculosis.

In the collateral form of pulmonary hyperæmia the indicatio causalis coincides with the treatment of the main disease. In obstruction in the lungs the indicatio causalis cannot be met. In disease of the heart, above all, in contraction of the mitral valve, the use of digitalis is to be recommended as a palliative until the heart's action have become retarded. The weaker the action of the heart becomes in the course of an asthenic fever, so much the more urgently are stimulants and nourishing food indicated. In like manner cause the position of patients with threatening hypostasis to be changed from time to time, in order to prevent a settling of the blood.

With regard to the indicatio morbi, bold venesection from a large opening is demanded in fluxion to the lung, arising from excessive cardiac action threatening life. The result here is astonishing. As soon as the volume of the blood has become lessened, the pressure diminishes in the arteries (as it depends upon two forces: first, the energy of the cardiac contractions; second, the fulness of the cavities of the heart). The patients often become able to breathe more freely, even during the operation, the bloody foam which they were expectorating vanishes, and life may be rescued from the greatest danger by aid of the physician. In cases like these, however, which have been called pulmonary apoplexy (*Lungenschlagfluss*), the danger arises with such lightning rapidity, that the physician usually arrives too late.

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* The very fine sweet grapes, which grow in the better vineyards of my present home, purge so strongly as to be inapplicable to the grape-cure. I have seen a severe diarrhœa set in after eating three to four pounds of Würtemberg grapes.

Collateral fluxion, also, when it threatens life, requires venesection. If, thereby, the force of the heart be diminished, the pressure, too, in the arteries of the hyperæmic parts of the lung is also reduced, the capillaries are less full, the transudation of serum, which was threatening, or had already set in, does not occur or ceases; and here, too, we often see the patient breathe more freely and more deeply, while the blood is yet flowing from the open vein. Since, however, in by far the greater number of cases, the venesection has an unfavorable effect upon the main disease by increasing the danger from exhaustion and impoverishment of the blood, let us not be led astray by these striking instantaneous results, so as to let blood without necessity, that is to say, unless life itself be threatened; but if, in the course of pneumonia, or pleuritis, or recent pneumothorax, with intense dyspnœa, a moist *râle* become audible, if the sputa become serous, etc., the danger is imminent; then pay no regard to the small pulse, or rather look upon it as a new reason for bleeding. The more recent the case, so much is collateral fluxion the more easy of recognition, and so much the more surely can we rely upon success.

Should symptoms of œdema threaten in the course of disease of the heart, immediate danger to life may demand a diminution of the volume of the blood, and the relief consequent upon venesection usually satisfies the expectation which has been entertained. In these cases, too, however, it is of the utmost importance to restrict blood-letting to the cases of the most urgent necessity. Persons with disease of the heart do not bear repeated venesection well; their blood, like that of emphysematous persons, and for the same reasons, after long duration of the disease, is poor in fibrin and albumen, and has great tendency to form serous transudations. Venesection, by diminishing the volume of the blood, renders it thinner; the original mass is soon reëstablished by absorption of liquid from the tissues and from the intestines; but the tendency to dropsical transudation and even to œdema of the lung is aggravated in this way.

In the other forms of hyperæmia of the lung which we have described, venesection is absolutely hurtful. Especially is this the case with hyperæmia occurring in asthenic fevers, no matter how great it may be, and though œdema threaten life. In these cases every thing depends upon our supporting the depressed energy of the heart, as by this means alone can its cavities be emptied, and blood flow away from the pulmonary veins. Blood-letting weakens the energy of the heart and augments the danger. As the latter class of cases are by far the most frequent, as they close the scene in almost all tedious and exhausting diseases, strong soups, fiery wines, camphor, musk, are much more frequently indicated for hyperæmia of the lung than is blood-letting. The difficulty of distinguishing between approaching heart-palsy and collateral fluxion in the course of pneumonia, which, from its great exudation, leads to intense fever, and at last also to enfeebled contractions of the heart, has been discussed above.

In addition to the procedures just spoken of, œdema of the lung may require the employment of emetics, for reasons already explained, as soon as the cough lacks energy, and the palsied bronchial muscles cease to aid in expelling the serous contents of the bronchi. Should the discharge of sputa be arrested, should the *râles* in the chest be increased, even if the patient cough, give an emetic of sulphate of copper or ipecacuanha, with tartar-emetic, but only when hope of saving life has not been extinguished. *Traube* recommends the use of acetate of lead—gr. j every hour—and the application of a very large blister to the chest, as a very efficient mode of treating œdema of the lung.

Œdema of the lung, as a symptom of general dropsy, demands the treatment of the main disease, but here, too, in the circumstances alluded to, an emetic may be indicated.

## *HÆMORRHAGES FROM THE RESPIRATORY ORGANS.*

In the majority of cases in which blood is coughed up, the bronchial mucous membrane is the source of the bleeding, but, as bronchial hæmorrhage is almost always the attendant or forerunner of disease of the lung, we have preferred to treat of this subject simultaneously with that of hæmorrhage from the pulmonary substance.

Under the head of hæmorrhage from the respiratory organs, accordingly, we shall discuss: 1. Bronchial hæmorrhage (broncho-hæmorrahie), by far the most frequent cause of hæmoptysis and pneumorrhagia. 2. *Hæmorrhagic infarction*, a hæmorrhage of the pulmonary tissue confined within narrow limits, and which causes no destruction. 3. *Pulmonary apoplexy proper*, an abundant hæmorrhage of the pulmonary tissue, caused by rupture of some of its large vessels, and causing destruction of the lung, with the formation of an apoplectic cavity.

Hæmorrhage from cavities and bleeding arising from the opening of an aneurism into the air-passages are to be treated of hereafter.

## CHAPTER VI.

### BRONCHIAL HÆMORRHAGE.

ETIOLOGY.—Wounds and erosions of the larger blood-vessels of the bronchial mucous membrane are extremely rare. Capillary hæmorrhages of the air-passages, too, are seldom of traumatic origin, or due to sloughing or ulceration of the membrane. As a rule, hæmorrhage proceeds from rupture of the capillaries, caused either by over-distention, or else by a morbid delicacy of their walls, a result of perverted nutrition. The trifling capillary hæmorrhages which occur in the first stage of acute bronchial catarrh, in cases of violent irritation of the air-passages, and in the circulatory disorder attending organic disease of the heart, proceed from the first of these causes, while in most of the hæmorrhages, in which large quantities of blood are poured into the bronchi, to be ejected thence by hæmoptysis or bronchorrhagia proper, they are due to the latter condition.

The fact, which has been too little appreciated hitherto, that nearly all bronchial hæmorrhages are mainly owing to a morbid state of the vascular walls—to a hæmorrhagic diathesis* of the bronchial mucous membrane—and do not depend upon over-filling of the vessels, is of great practical importance. The truth of this is shown by the fact, not only that attacks of hæmoptysis and bronchorrhagia are not usually preceded by bronchial hyperæmia, but that the spitting of blood almost always persists, and, in fact, often does not assume an obstinate character until after the patient has lost a good deal of blood, so that his vascular system is considerably depleted.

A tendency to abundant bronchial hæmorrhage—to a hæmorrhagic diathesis, according to the above definition—1. Is met with in rare instances, occurring unexpectedly in young persons, apparently in blooming health, and of vigorous constitution. In such cases, we as yet have absolutely no explanation of the disorder, which is usually limited to the capillaries of the bronchial mucous membranes, and which is often followed by such sad results.

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* We employ this general term merely to signify a morbid tenderness of the vascular walls, and not a morbid condition of the blood; although we admit that the latter may sometimes so modify the nutritive state of the walls of the blood-vessels as to impair their resisting power, and thus to lead to a hæmorrhagic diathesis.

2. Much more frequently we find similar tendency to profuse capillary hæmorrhage from the bronchi in young people, between the ages of fifteen and twenty-five years, of delicate health, and having marked weakness of constitution. Such patients frequently have been orphans from an early age, having lost their parents by consumption. They have suffered from rickets, or scrofula, in infancy, have often bled at the nose, and have rapidly grown tall, without at the same time acquiring any corresponding development of the various organs of the body. Their long bones are thin, their chest narrow, and even their skin seems unusually delicate and transparent; their cheeks redden easily, and blue veins may be traced over the ridge of the nose and the temples. One might almost be tempted to attribute the remarkable frequence of bronchial hæmorrhage in persons of this type to a deficience of vital material, which, having been immoderately expended during the maladies of childhood, and by the rapidity of the growth, has proved insufficient to maintain normal nutrition of the capillary walls, just as we are accustomed to ascribe the occurrence of spontaneous bleeding after severe illness, tedious suppuration, or great loss of blood, to a kindred source of exhaustion of the nutritive principle. Such an hypothesis, however, does not explain why the seat of hæmorrhage should first be in the nose, and afterward in the bronchi, and why hæmorrhage scarcely ever occurs into the brain, or into other organs, in patients of this class.

3. There is a great predisposition to capillary hæmorrhage from the bronchi in persons suffering from tuberculosis and consumption. The frequence of abundant hæmorrhage in all stages of these diseases arises partly because individuals who are liable to such bronchial bleeding are equally liable to tuberculosis, and to consumption of the lungs, and because the tendency to bleed does not cease when the lungs become affected, and partly because deposit of tubercle and chronic inflammation cause the pulmonary tissues and the bronchial mucous membranes to become relaxed, so that the capillaries which are imbedded in the relaxed tissues (now no longer capable of resisting their undue dilatation) suffer excessive distention and attenuation of their walls, whereby they become more easy of rupture.

Finally, coalescent masses of tubercle and centres of inflammation, by compression of vessels, give rise to fluxionary and obstructive hyperæmia, by which rupture of the capillaries is favored.

Prejudice in favor of the narrow views of *Laennec* and a belief in the ancient Hippocratic theorem, *Epi aimatos emeto phthoe kai tou puou katharsis ano*, have seriously biased the judgment of physicians as to the relation between bronchial bleeding and pulmonary tuberculosis, and have given rise to extravagant and erroneous ideas. Many physicians do not hesitate to accept a brisk hæmoptysis as a sure sign of incipient, or even of established tuberculosis, although the patient may present no symptoms, either subjective or objective, of disease of the lungs, and when, soon after the occurrence of hæmoptysis, signs of consumption have arisen, they confidently assume that the bleeding has been caused by the presence of tubercle, or by the process of its deposit in the lungs.

I must earnestly protest against this opinion, as altogether unwarranted, and fraught with danger to the patient. Cases undoubtedly occur, in which tubercles and inflammatory processes form in the lungs, in a manner so latent that no tokens of the disease are manifested by the individual affected, until he is suddenly attacked by a fit of hæmorrhage. Such instances, however, are exceptional.

In the very great majority of cases in which the first attack of hæmoptysis has not been preceded by either cough, dyspnœa, or other sign of pulmonary disorder, the lungs are free, and by no means the seat of tubecular deposit, at the commencement of the bleeding.

It is true that such subjects rarely die of hæmorrhage, so that we do not often have an opportunity of examining their condition *post mortem.* However, if we collate the reports scattered through our literature and compare their statements, we shall assure ourselves that they substantiate the correctness of the above remarks. I have repeatedly failed to find *post-mortem* traces of pulmonary tubercle, or of any other destructive disorder in the lungs of individuals who have died suddenly of pneumorrhagia, while in enjoyment of apparent health.

That bronchial hæmorrhage is by no means so rare an event, where there is no grave disease of the lungs, is shown, moreover, by the tolerably numerous cases in which persons, after suffering one or more attacks of pneumorrhagia, regain their health completely, and indeed often live to an advanced age, and after death present no discoverable traces of extinct tuberculosis in their lungs.

That bronchial hæmorrhage, as a rule, should precede the disease of the lung, in the cases where the initial signs of consumption follow immediately upon an attack of hæmoptysis is also strongly in contradiction of the theories of *Laennec*, to which, nevertheless, most modern physicians adhere without question. According to *Laennec's* view, there is but one kind of consumption—tubercular consumption. "As bronchial hæmorrhage can never produce a deposit of tubercle, all genetic connection between such hæmorrhage and the consumption must be denied absolutely. Hence, where the first symptoms of consumption follow close upon a hæmoptysis or pneumorrhagia, we may assume confidently that the tubercular deposit has formed either simultaneously with or prior to the occurrence of the bleeding." Such argument, though logical, is fallacious, because based upon the erroneous hypothesis that consumption of the lungs always arises from tubercular deposit. Unbiassed and careful observation of patients, who, without warning and often in the midst of exuberant health, have been attacked by pneumorrhagia or hæmoptysis, and who, without rallying, have perished in a few months of a phthisis florida, a "galloping consumption," has taught me that such patients scarcely ever succumb to a pulmonary tuberculosis in its stricter sense, but that they usually die of a form of consumption as yet but little thought of, and of which bronchial hæmorrhage is the immediate cause, *Laennec* to the contrary notwithstanding. When, after a bronchial hæmorrhage, coagulated blood is retained in air-vesicles and bronchi, its irritating effect is quite as great upon surrounding parts as is that of a thrombus or coagulum within a vein upon the vascular tissues.

The bronchitis and pneumonia arising from such a source may result in various ways. (See below.) A very common consequence is, that both clot and inflamed pulmonary tissue undergo a caseous metamorphosis, with subsequent decay. These pathological and anatomical processes agree closely with the type which consumption assumes when it immediately follows a bronchial hæmorrhage in an individual previously vigorous and healthy, and proves fatal in the course of a few months.

Finally, I may observe that the bronchial hæmorrhages which occur in an established case of consumption also cause chronic pneumonia and destruction of the tissues, and thus hasten the fatal termination. The fact that the occurrence of hæmoptysis in the course of a pneumonia is a serious event, and that the disease often rapidly grows worse immediately afterward, is generally admitted by physicians, although, as a rule, it has been falsely interpreted; it being a common supposition (but one which is rarely the true one) that fresh tubercles have formed, which, by some, are thought to have caused the hæmorrhage, and by others to have accelerated the consumption.

As my opinions regarding the relations between bronchial hæmorrhage and pulmonary consumption differ in some respects from the prevailing views upon this subject, I propose briefly to state them in the following paragraphs:

1. Bronchial hæmorrhage occurs oftener than is generally believed, in persons who are not consumptive at the time of the bleeding, and who never become so.

2. Copious bronchial hæmorrhage frequently precedes consumption, there being, however, no relation of cause and effect between the hæmorrhage and the pulmonary disease. Here both events spring from the same source—from a common predisposition on part of the patient both to consumption and to bleeding.

3. Bronchial bleeding may precede the development of consump-

tion as its cause, the hæmorrhage leading to chronic inflammation and destruction of the lung.

4. Hæmorrhage from the bronchi occurs in the course of established consumption more frequently than it precedes it. It sometimes, although rarely, appears where the disease is as yet latent.

5. When bronchial hæmorrhage takes place during the course of consumption, it may accelerate the fatal issue of the disease, by causing chronic destructive inflammation.

ANATOMICAL APPEARANCES.—Upon *post-mortem* examination of those who have died of bleeding from the bronchi, the air-passages are found more or less extensively and completely filled up with masses of clotted blood. Sometimes the mucous membrane has a uniform dark-red stain, from effusion of blood into its tissues, and it is swollen, relaxed, and bleeds upon pressure. In other cases, again, the entire contents of the capillaries seem to have been discharged, the mucous membrane presenting a pale and bloodless appearance. The source of bleeding is never found to be of the nature of a mechanical or ulcerative lesion.

The lungs, at points where the blood has descended into the air-vesicles, are heavier, denser, and more or less reddened. If the bronchi remain filled with their bloody contents, escape of air from the air-cells is prevented, and the lungs remain inflated when the chest is opened. Where death has been caused by hæmorrhage, there is extreme anæmia of all organs.

In cases where death has taken place some time after the hæmorrhage has ceased, either no trace whatever of the former bleeding is found in the lungs—and, indeed, this is most commonly the case—or else the signs are found of chronic inflammation in its different stages, which, however, is never to be ascribed to the hæmorrhage, unless a greater or less amount of broken-down blood-clots, in a state of fatty degeneration, be also found in the bronchi. I have published a case from my clinique, in which the *post-mortem* appearances exhibited the entire process, in the most striking manner, in which coagula, bearing a perfect resemblance to old thrombosis of the veins,* were found in the bronchi.

SYMPTOMS AND COURSE.—The admixture of small quantities of blood in catarrhal expectoration—occurring in the form of minute streaks traversing the mass—is a very common and quite harmless symptom. The expectoration of a somewhat larger amount of blood—either pure or mingled with bloody mucus—which sometimes follows upon the inhalation of acrid vapors, or after other severe irritation of the air-passages, and is of still more frequent occurrence in disease of the heart, from obstructive hyperæmia, seldom results seriously, and rarely imperils the patient's life.

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* "Upon the relation of bronchial and pulmonary hæmorrhage to pulmonary consumption." Inaugural dissertation of Doctor Bürger. Tübingen, 1864.

Very profuse hæmorrhages, of a very different nature, often arise from a morbid inability of the capillary walls to resist the pressure of their contents, and it is to these that we usually allude, when we employ the terms "spitting of blood" (hæmoptysis) and "bursting of a blood-vessel" (pneumorrhagia). In such cases an observant physician may long foretell the occurrence of a hæmorrhage in patients of the constitutional habit above described, especially if they have often bled at the nose, and have suffered now and then from palpitation of the heart, and oppression of breathing. It is but occasionally, however, that the attack itself is preceded by warning symptoms or by sensations of constriction of the chest. Far more commonly the long-dreaded hæmorrhage sets in suddenly. The patient feels as though a warm liquid were oozing up from beneath the sternum; he perceives a strange sweetish taste in his mouth, and, upon attempting to clear the throat, finds that he expectorates pure blood or bloody mucus,—"that he is raising blood." Such a discovery generally has a very depressing effect, even upon individuals of the utmost courage. The saying of Mephistopheles, "Blood is a quite peculiar juice," stands out here in its full reality. Though the bleeding may have been trifling, yet we often find the patient tremulous, pale, and almost fainting. Soon after "raising" the first blood, a sense of titillation induces inclination to cough. Coarse, moist *râles* and a gurgling sound are audible in the chest; a short, full, loose cough follows, and frothy, bright-red blood gushes from the mouth, and often, too, from the nose. Short pauses intervene between the coughing-fits, during which more blood seems to be escaping and collecting in the tubes, and, in this manner, large quantities of it are often ejected in a short time. (The quantity of blood lost may vary from an ounce or two to a pound or more.) The attack may subside in course of half an hour, sometimes sooner; at times not for several hours. The mucus continues to retain a bloody stain, or is mixed with blood, but the blood is no longer pure. Attacks of hæmoptysis are rarely solitary, however. They almost always recur in course of a few hours, or perhaps the next day "in spite of the most careful treatment!" Indeed, the attacks are generally repeated for two or three days, or even a week, until at length the patient, who has grown pale and feeble, obtains a respite from his hæmorrhage, which may last for months or even years.

In such cases, and, indeed, in most others, the course of bronchial hæmorrhage is singularly uniform, whether it occur in consumption, or attack persons whose lungs are exempt from tubercle or any other known disease.

Very rarely is life directly endangered. It is important that we should bear in mind that patients nearly always survive the attack, in spite of intense prostration, tendency to syncope, and other signs of impending dissolution. Death from bronchial obstruction and impeded respiration is somewhat more common than death from hæmorrhage. Physical examination of the chest gives negative results, with the exception of a few coarse, moist *râles;* and it is both useless and imprudent to agitate the patient by constant and inconsiderate percussion and auscultation. If blood enough pass into the vesicles to expel the air from any considerable portion of the lung, the percussion-sound over that point is flat and dull, and the respiratory murmur either feeble and indistinct, or else bronchial.

In many cases the patients, after expectorating small masses of bloody mucus, and of clotted blood, for a while, recover rapidly. If blood have lodged in a bronchus, so as to close it and render it impermeable to air, its color is no longer bright red, but grows dark, inclining to black.

In most patients, and even in those who soon regain their health after "a hæmorrhage," by attentive observation, during the few days immediately following the bleeding, we shall discover a more or less violent inflammatory condition of the lungs and pleura. I at least, ever since my attention has been drawn to the occurrence of this consecutive pleuro-pneumonia, have almost always succeeded, two or three days after an hæmoptysis, in finding an elevation of temperature, and increase in the frequence of the pulse, constitutional disturbance, and lancinating pains of more or less severity in the sides of the chest. Moreover, I have frequently found a slight dulness, or a friction sound, with subcrepitant *râles.* Even in cases where considerable time had elapsed since the hæmorrhage, I have usually been able to discover, by careful examination, that, immediately after the occurrence of the bleeding, symptoms, more or less distinct, had arisen, of inflammation of the respiratory organs. I cannot comprehend why these sequelæ of bronchial hæmorrhage, which are almost constant, should hitherto have attracted so little attention, and why they are hardly anywhere mentioned in books on the subject.

The most frequent termination by far, of this consecutive inflammation, is *resolution.* The symptoms often vanish in a few days, and the patient becomes completely convalescent.

In other instances the elevation of temperature and increased frequence of pulse continue. The general health is also influenced by the persistence of the fever. The pain in the chest, too, continues in a mild form, and is generally ascribed, by the patient, to rheumatism. The respiration remains hurried, and the patient coughs, expectorating a muco-purulent sputum. If, besides these signs, we find dulness upon percussion at some point in the chest, the respiratory murmur feeble, or indistinct, if the patient manifestly be growing thinner and more miserable, we shall have very strong reason to fear that a destructive process has been set up in the lung, and that the patient will die of phthisis; nevertheless, all hope is not to be abandoned. In many cases, after a few weeks, the fever, the pain, the dyspnœa, the cough, and the expectoration, all subside, the patient "feels as though he had had a severe fit of sickness." His recovery is rapid and complete. Physical examination shows a depressed spot in the thorax, in the neighborhood of which percussion is somewhat deadened and flat, while the respiratory murmur is enfeebled. The pneumonia has resulted in wasting and contraction of the inflamed portion of the lung. In the dissertation before referred to, two cases of this kind (one of which concerned a former assistant of mine) are carefully detailed, and, since then, I have ascertained, by a large number of observations, that such a result is a very common one.

If a chronic pneumonia, proceeding from profuse bronchial hæmorrhagè, do not take a turn for the better; if the patient, on the contrary, fail more and more under the effect of intense fever with evening exacerbations, and profuse night-sweats; if the sputa become more copious and purulent; if physical evidence of the formation of caverns arise, we may conclude that the chronic pneumonia has terminated in *cheesy metamorphosis* and *disintegration of the inflamed pulmonary tissue.*

I may finally repeat that persons, who have suffered a severe hæmorrhage from the lungs, éven though it may not have been followed by any ill effects, and although they may have recovered from it entirely, are, nevertheless, in danger of dying, sooner or later, of pulmonary tuberculosis, or of pulmonary consumption.

DIAGNOSIS.—Hæmorrhage from the bronchi, not unfrequently, is confounded with epistaxis, particularly if the latter proceed from the posterior nares, or if the patient lie upon his back during the bleeding. Here the blood flowing into the pharynx reaches the larynx, and is then frequently coughed and hawked up, to the great terror of the patient and his relatives. Long before the physician makes his appearance, the regulation doses of salt and vinegar have been administered, and it is of importance, in order that he be not deceived himself in the midst of the general consternation, that he should deliberately inspect nose, gums, and palate, and inform himself precisely whether the patient have not bled at the nose on the previous evening.

The distinction between hæmoptysis and hæmorrhage from the stomach may also have its difficulties, particularly if we have to decide upon the source of a hæmorrhage which has taken place years before.

In hæmoptysis, the irritation of the cough often provokes retching and vomiting, or the blood may be swallowed, and afterward thrown up. Conversely, violent hæmatemesis is almost always attended by coughing, small quantities of blood getting into the larynx; hence, the patients are not always able to tell, exactly, whether they have coughed up the blood or vomited it up. In treating of hæmorrhage of the stomach, we shall enlarge more fully upon the distinction between the two conditions, and merely remark, that we must, first of all, inquire whether the cough have been followed by vomiting, or the vomiting by cough; secondly, that we must accurately ascertain whether cardialgic distress have preceded the gush of blood or not; thirdly, examine carefully as to whether the bleeding have been followed by black, tar-like stools, or whether the patient have voided mucus tinged with blood for a few days after the attack. If, moreover, we have opportunity to examine the blood which has been discharged, that from the air-passages is usually bright red, frothy, with alkaline reaction. Should a clot form, it will be soft, and specifically light, as it contains bubbles of air. On the other hand, blood which has been vomited is dark, and even black, excepting where a great artery of the stomach has been eroded. It is not mixed with air-bubbles, but contains remains of food; its reaction is usually acid, and the clot, if it forms one, is firm and heavy.

We have now to add a few words regarding the distinction of hæmorrhage of the bronchial capillaries from the bleeding arising from a wound in the tissue of one of the larger vessels, which traverse the walls of a cavity. It is admitted, by some authors, that a hæmorrhage of moderate degree, a hæmoptysis, proceeds generally from the capillaries of the mucous membrane, but that all profuse bleeding, amounting to a pneumorrhagia, springs from rupture, or erosion of larger vessels. So convinced are they of the justness of this view, that they assume that any one who has had a violent hæmorrhage, be he never so healthy in appearance, has cavities in his lungs, which have heretofore escaped observation. The objection, that so large an effusion of blood cannot possibly flow from the bronchial capillaries, is untenable; since capillary hæmorrhages from the nasal mucous membrane are often so profuse as to endanger life, and a hæmorrhage from the bronchi of equal activity, if it flow from a sufficiently large surface, may very easily yield so much blood as to fully warrant application to it of the term "bursting a blood-vessel," instead of "raising blood." Moreover, many people who have spit blood are prone to exaggeration, and talk of "gushes of blood," and of "coughing up blood by the pint," while the actual amount lost has not been nearly so large. Besides, it is highly improbable that there should be undiscovered cavities in the lungs of all persons who have suffered from severe and profuse bleeding from the air-passages, but who, in other respects, seem to be in good health; and it would be very extraordinary if hæmorrhage from small, latent cavities were to be of much more frequent occurrence than from large and recognizable ones. But we have direct proof that the blood lost in pneumorrhagia does not come from a large vessel; at all events, not from a branch of the pulmonary artery. According to the classical picture of *Rokitansky*, the branches of the pulmonary artery, as a rule, soon become obliterated in the various forms of consumption. Sometimes, however, they become perforated by erosion, or suffer rupture. *In them runs the most venous and darkest blood of the entire body.* Now, in almost every case, not only of hæmoptysis, but of pneumorrhagia, the blood is of a remarkably bright-red color, so that, in the differential diagnosis between hæmoptysis and hæmatemesis, great stress is laid upon the light color of blood which flows from the lungs and air-passages. (See above.) It is only when large quantities of dark blood are ejected, that we are justified in inferring that a branch of the pulmonary artery has become eroded or ruptured. A striking example of this kind occurred in my clinique, and has been made public (*see Bürger's treatise*). Such accidents, however, are extraordinarily rare, in comparison with the frequence of hæmorrhages of bright-red blood. This bright-red blood can only come from the bronchial mucous membrane; or, at all events, either from a branch of the bronchial artery or of the pulmonary vein.

PROGNOSIS.—The prognosis, as regards immediate danger to life, is, as we have shown, on the whole, favorable, in spite of the alarming character of the symptoms. The prognosis, however, as to complete recovery, is exceedingly bad. The slighter the provocation, the less apparent the cause of the hæmorrhage, so much the graver is the omen. The prognosis is better when rupture of the capillaries has been caused by excessive hyperæmia, due to direct injuries, excessive action of the heart, or other serious irritants, provided that the irritation thus set up can be allayed. Suppressed menstruation and repressed hæmorrhoids can only be counted among these causes with extreme reserve, much as the patients may be inclined to attribute their blood-spitting to such anomalies, and readily as they may become satisfied, when the physician partakes in their belief. Absence of the menses is much more often the consequence of the disease than the cause of it, and the same holds good for any hæmorrhoidal bleeding which may have existed prior to the attack, and which has ceased during or immediately after it.

TREATMENT—*Indicatio Causalis.*—If excessive hyperæmia of the bronchial mucous membrane play a material part in the origin of a bronchial hæmorrhage, or if it be attributable solely to increased lateral pressure upon the capillary walls from within, the indicatio causalis may, in such cases, but only in such cases, demand venesection. In most cases, lateral pressure has but little to do with the bleeding. It does not cease, though the pressure be relieved, the vessels empty, and the patient be almost dead from hæmorrhage. Let us but call to mind those waxy, pallid sufferers from epistaxis, whose nostrils we often have to tampon, in order to master the bleeding; and our lancet will stay in its case as long as the heart's action is moderate. Indeed, we must restrict blood-letting to cases where, in spite of the bronchial hæmorrhage, there is a persistent and alarming hyperæmia of the lung.

Since we are unable to assign a reason for the delicacy and thinness of the capillary walls, which is the chief source of bronchial bleeding, we are forced to admit, that the indicatio causalis cannot generally be met—that it is not in our power to combat the hæmorrhagic diathesis by any rationally specific means. At all events, it is scarcely possible, after hæmoptysis has set in, to effect any rapid change in the abnormal state of the capillary wall. It is preferable, in dealing with patients threatened with this affection, or who have already suffered an attack, to preserve them with peculiar care from all hurtful agents which could injure their nutritive condition. We should order simple, unexciting, nourishing food; moderate bodily exercise in the open air; should carefully regulate the action of the bowels; should prohibit all excess *in baccho et venere*, and enjoin avoidance of mental excitement. Where there is a decided want of red corpuscles in the blood, the exhibition of the milder preparations of iron, the employment of Pyrmont-water, or that of Driburg or Imnau, are to be recommended, and the neglect of these measures is a gross blunder.

The *indicatio morbi*, above all things, demands a cautious regimen. We should, in the first place, seek to calm the spirits of the patient, which are always much excited; and, inasmuch as these attacks are almost always repeated several times, it is well to save him from further agitation, by straightway informing him that there is more blood to come, while, at the same time, we should absolutely deny the possibility of his bleeding to death. Indeed, we are certainly warranted here in deceiving the patient, by affecting to make light of the affair, and even to represent the hæmorrhage as a salutary process.*

With a little tact, the physician may leave his patient in a state of comfort and peace of mind, whom he has found in the most painful uneasiness—a success of no slight importance. Take care that the chamber be cool; forbid all hot drinks, and let all food be eaten cold. Interdict all conversation, and make the patient stoutly resist the provocation to cough. Coughing in hæmoptysis is quite as hurtful as is snuffling and wiping the nose in epistaxis. Finally, remove all portions of the clothing which press upon and confine the chest, and cause the patient to assume a half-sitting posture in bed. The most powerful means of combating the bleeding is the use of cold. We apply this in the form of cold compresses, and, when the bleeding is very severe, in the shape of frozen compresses.*

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* I here call to mind the effect of conjuration and penance on the spirits, and indirectly upon hæmorrhage.

In addition to this, let him swallow small pieces of ice, or give small doses of ice-water; or we may apply the cold in the form of clysters, to which, from time immemorial, a little vinegar has always been added. Besides cold, a number of substances have the reputation of arresting hæmorrhage without our being able, physiologically, to explain how they act. Under this head, before all others, come two remedies: common salt, and the acids, which, curiously enough, when taken in excess, occasion a scorbutic state of the blood, a bad nutritive state of the capillaries, and lead to hæmorrhage. However this may be, we must make the patient swallow one or two spoonfuls of finely-powdered, dry salt. Sulphuric, or phosphoric acids, are still more preferable, especially the elixir acidum Halleri, of which we give ten drops every two hours, mixed in a sufficient quantity of water. A series of other hæmostatic remedies follow these, which are not of such generally acknowledged efficacy as the acids, and which, being less innocent, are therefore less highly esteemed. Among these is acetate of lead, of which the English physicians maintain that, for internal hæmorrhage, there is "nullum simile aut secundum." Next come secale cornutum, oleum terebinthinæ, balsam copaivæ, ratanhy, and other medicines.

*Wunderlich* particularly recommends the exhibition of secale cornutum, in doses of from five to ten grains, until a prickling and numb sensation in the fingers sets in. A formula, much in use in very obstinate hæmoptysis, is—℞. Balsam copaiv., syrup balsam, aquæ menth. piper, spirit. vini rectif. āā ℥ j; spirit. ether nitrici ʒ ss; ♏. S. ʒ ij every two to four hours. These various drugs are only to be made use of in very dangerous cases, and we should not forget how impotent all these styptics are in severe bleeding of the nose, where, moreover, we are able to apply them directly to the bleeding point. Latterly, inhalations of a solution of sesquichlorate of iron (℈ j to ʒ ss with ℥ vj) has been recommended as exceedingly serviceable against hæmoptysis. The most alarming hæmorrhages are said to have been arrested, by this means, in the course of four or five minutes. My own experience does not confirm this recommendation. The narcotics should be employed freely. The more restless the patient, the more violent his cough, so much the more boldly should we order opium. Let a Dover's powder be taken at night, and during the day an emulsion, with half a drachm of laudanum, or half a grain of morphine.

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* Fill a tin or copper warming-pan with ice, salt, and water, then lay it upon a well-squeezed wet compress, the moisture of which soon freezes. These compresses are greatly to be preferred to the heavy bladders of ice.

## CHAPTER VII.

### PULMONARY HÆMORRHAGE WITHOUT LACERATION OF THE PARENCHYMA—HÆMORRHAGIC INFARCTION—METASTASIS TO THE LUNGS.

In former editions of my text-book I have treated of pulmonary hæmorrhagic infarction, which occurs from disease of the heart, and the so-called metastatic infarction in separate chapters, since, notwithstanding the complete identity in their essential anatomical lesions, the difference in their extent and seat, and, above all, the different manner in which they originate, seemed to me to demand it. But from an opinion of *Rokitansky*, from an excellent essay by *Gerhardt*, and especially owing to a series of observations of my own, published in the dissertation of Doctor *Hopf*,[*] I have become satisfied that my former views were erroneous; that the variations in magnitude and in the seat (neither of which are constant) constitute no real difference, and that the modes of origin of hæmorrhagic infarction in heart-disease, and of that of metastatic infarction from thrombosis of a vein, or from external suppuration, or sanious ulceration, are identical.

Etiology.—Hæmorrhagic infarction consists in a capillary hæmorrhage, confined to a small and sharply-defined section of the lung, and often bounded by the limits of a single lobule. The blood is effused, partly within the cavity of the vesicles and terminal bronchi, and partly lies in their interstices between the fibres of elastic tissue by which the air-cells are entwined. The hæmorrhage does not produce laceration of the lung-substance. The abrupt boundary of a hæmorrhagic infarction is caused by the fact that the bleeding only comes from the capillaries pertaining to a single twig of the pulmonary artery. The range of the capillary system of an artery depends upon its size; hence hæmorrhagic infarctions which arise within the capillary limit of a large branch of the pulmonary artery are far more extensive than one which forms about a smaller twig. As the main trunks of the pulmonary artery enter the roots of the lung in company with the great bronchi, and ramify toward the surface, constantly growing smaller by repeated subdivision until each ultimate twig terminates in a single lobule, the reason is plain why the infarctions occurring in the interior of the lung are large, and why peripheral infarctions preserve both the size and the cuneiform shape of the superficial lobuli. Upon careful examination of an arterial branch within whose range a hæmorrhagic infarction has formed, we find in it a clot by which its calibre is more or less obstructed. This is easily demonstrated in the larger vessels, but in the very small ones it is sometimes difficult.

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[*] Zur Diagnose des Hæmorrhagischen Infarctes. Inaugural Dissertation von Dr. Hopf. Tübingen, 1865.

That the obstructing coagulum has not formed at the place of its lodgment, but that it comes from some remote region of the body, whence it has become detached and swept into the current of the blood, until, finally, it has become impacted in some branch of the pulmonary artery too narrow to admit of its passage, has long been recognized as the conditions under which hæmorrhagic infarction arises. The credit of this valuable discovery is due to *Virchow*. That investigator, by introducing particles of fibrin, muscle, elder-pith, and the like into the jugular veins of dogs, demonstrated by dissection that these foreign bodies blocked up branches of the pulmonary artery, and produced hæmorrhagic infarctions, lobular pneumonia, and small abscesses, beyond the points obstructed. Conversely, he proved by dissection of bodies, in which the diseased spots so long known as metastases had been found, that the arteries leading to the affected points were occluded by an *embolus*—a fibrinous plug, which undoubtedly had proceeded from a *thrombosis* of a superficial vein, or from particles whose origin was indisputably traceable to some region of suppurative or sanious ulceration upon the surface.

Of late, the doctrines of pyæmia and of septicæmia have undergone many revolutions; but that of embolism—that is, of the dependence of hæmorrhagic infarction upon the introduction of clots, or of particles of tissue into the circulation—has remained unshaken.

It is easy to understand why metastatic infarctions of the lungs are caused by emboli from disintegrating thromboses of peripheral veins, or from suppurating or sanious surfaces. When an embolus is detached from its point of origin by the current of the blood, it meets with no obstacle on its way to the heart, as the veins through which it travels are constantly growing larger. It passes unhindered into the right heart and into the pulmonary artery, and is not arrested nor impacted until it arrives at some branch of the latter whose diameter is less than its own. Upon similar grounds, it is the rule for emboli, which originate from the roots of the portal vein, or which enter the portal vein in cases of ulceration or of sanious discharge from the intestines, to pass into the ramifications of the portal vein within the liver, causing metastases in that organ, and for emboli which come from the lungs on the left side of the heart to occlude the arteries of the spleen, kidneys, or brain. Where exceptions to this rule occur, as when we sometimes find infarctions in organs whose arteries an embolus could not have reached, without first passing through the capillaries of another organ (for instance, infarction of the liver in thrombosis of a peripheral vein), it seems probable that the embolus at first has been minute, but that during its course through the system it has grown larger by accretion of fibrin. The very common occurrence of hæmorrhagic infarctions after injuries of the skull, where the diploë have been penetrated, is simply due to the gaping of the walls of the veins of this region, which, being adherent to the tables of the skull, are prevented from collapsing, so that the entrance of coagula into them of course is facilitated.

In the hæmorrhagic infarctions which so often arise in diseases of the heart, especially in cases of disease of the mitral valve, the existence of clots in the arteries leading to them has long been known. But the explanation generally has been that the escape of blood into the vesicles and their interstices has compressed the capillaries and prevented the outflow of blood from them, and that in consequence of the stagnation so produced the arterial contents have coagulated. This was formerly my opinion, although I could not ignore that the extreme obstruction of the blood in the pulmonary circulation, to which I ascribed the infarction in disease of the heart, did not at all account for the restriction of the capillary hæmorrhage to separate and abruptly-defined sections of lung. I am now convinced that, in disease of the heart, hæmorrhagic infarction also arises from embolism, as has been proved by *Rokitansky* and *Gerhardt*. The emboli which block the artery in disease of the heart do not come from the greater circulation, like the emboli which produce metastatic infarction, but from the right side of the heart, especially from the right auricle, in which clots usually exist firmly entangled in the trabeculæ, and which are one of the results of the sluggishness of the circulation. If a particle of this clot be torn off and washed away by the current of the blood, a branch of the pulmonary artery becomes obstructed by it, and hæmorrhagic infarction ensues. The fibrinous coagula thus detached from cardiac thromboses are generally larger than those which come from the aortic circulation. We thus find a very simple explanation of why the infarctions of heart-disease are more extensive than metastatic infarctions, as well as of why the former are often found in the interior of the lung, near its roots, while the latter are generally situated near the periphery. As very minute particles also may be washed away from the thromboses of the right heart, we likewise see how, besides the larger infarctions at the roots, smaller peripheral ones also occur in heart-disease.

The process still remains to be explained by which obstruction of an afferent arterial branch produces capillary hæmorrhage in the region about the obstructed vessel, a process which, at the first glance, seems by no means easy of elucidation. The theory of *Rokitansky*, "that occlusion of the minutest arterial branches of the lung and its capillaries causes a collateral hyperæmia, which results in hæmorrhage and exudation," is unsatisfactory to me, for the bleeding does not proceed from neighboring capillaries, but from those of the obstructed vessel. Nor does *Virchow* give a sufficient explanation of the capillary hæmorrhage. It is, therefore, all the more welcome and interesting that *Ludwig* has furnished a complete and final solution of the problem, from an entirely unbiassed point of view, by showing the influence which the contraction of an artery has upon its capillaries. His words are as follows: "Tension within the artery below the point of constriction is diminished, since a liquid flowing through a narrow tube loses more of its impetus than in flowing through a wide one. But we must not infer from this that, when an artery is constricted, the contents of its capillaries are lessened, and that the parts which they traverse grow paler. The sluggishness of the stream thus produced in the capillaries rather has the *effect of allowing the heavy blood-corpuscles to collect and become crowded together;* now, as two or more blood corpuscles, if brought into contact, are apt to become permanently adherent, the blood itself can form a plug capable of closing the capillaries. Such an occurrence, which converts the capillaries into blind appendices to the artery, must cause an increase in its internal pressure." Let us add that in consequence of the pressure, which, after the stoppage, is as severe in the capillaries with thin, delicate walls, as in their afferent vessels, a rupture of the distended walls and an escape of blood ensue. This furnishes a simple and entirely satisfactory explanation of the origin of the hæmorrhage, as well as of its limitation to the region supplied by the occluded artery. The utmost that can be advanced against the accuracy of this explanation is, that the artery leading to an infarction not only is narrowed, but is entirely closed. Such an objection, however, is untenable. The infarction of an embolus, which we find usually takes place at the bifurcation of an arteriole, very rarely produces absolute closure of it at first, but merely causes in it more or less obstruction. Afterward, when the infarction has become established—an event requiring but little time—fibrin is deposited upon the embolus, and closure of the vessel becomes complete.

Under conditions similar to those under which metastatic infarctions are observed, we sometimes find in their stead, or accompanying them, *circumscribed* pneumonic infiltration and small abscesses. As a rule, these appearances are manifestly the latter stages, the products of infarction; and it is not surprising that such products should be very common, and should form very rapidly, when the emboli consist of fragments of sanious or gangrenous tissue, capable of most pernicious action upon the parts in contact with them. The truth of this idea is supported by the fact that in disease of the heart, where the emboli are simple fibrinous clots, which are much less dangerous to the parts adjacent to them, destructive pneumonia and the formation of abscess are far more rare. The inflammatory reaction which occurs in the latter form is generally more of a nutritive than of a destructive character, and often results in a development of connective tissue, by which the infarction becomes incapsulated.

In a few instances, both formation of abscess and circumscribed pulmonary gangrene seem to ensue, the latter being a rare termination of infarction. Here the extravasation and compression of the capillaries above described cause secondary coagula to form, and this time it is in the nutritive vessels of the lung, the ramifications of the bronchial arteries, the pulmonary "*vasa privata.*" Nutritive material is thus withheld from the point of infarction, which dies and putrefies, or becomes gangrenous.

Anatomical Appearances.—We rarely find the blood liquid in dissection of recent infarctions; as a rule, it is coagulated. This circumstance is easy to account for, if we reflect that the locality impedes a discharge of the blood, and that, if the patient survive the attack for any length of time, the liquid part is absorbed, while the coagulable portion is retained. The blood is easily expelled from the bronchi by coughing, by the action of the bronchial muscles, and by that of the ciliated epithelium, but forced expiration can only empty the vesicles in part, and they have no muscles nor ciliary epithelium.

Hæmorrhagic infarctions which occur in disease of the heart generally vary in size from that of a hazel-nut to that of a hen's egg. They are of a blackish-red or blackish color, completely inelastic, and void of air, so that they can be felt from without like hard knots. Their cut surface presents an irregular, coarse, granulated aspect, from which a brownish-black mass may be scraped off with the scalpel. In the immediate vicinity of this sharply-defined spot the lung is usually full of blood and œdematous from collateral fluxion. Its seat, as already mentioned, is usually at the middle of the lower lobes, or near the roots of the lungs; more rarely at the surface. Microscopic examination shows the capillaries to be distended by blood-corpuscles, which are also collected in the tissue outside of the capillaries.

Where the infarction is of long standing, it looks paler and yellowish, the fibrin having undergone fatty degeneration, and the coloring matter of the blood being partially decomposed. Still later, the fatty fibrin is absorbed, and part of the hæmatin has turned into pigment, and the only remaining trace of the infarction is a blackish induration in the lung. In the rare instances in which an abscess forms it may become incapsulated, and its contents may thicken into a cheesy or calcareous mass.

Gangrene of the lung, as a result of hæmorrhagic infarction, will be described in Chapter XII.

In explaining the pathogeny of metastatic infarction, we have already alluded to the small volume, the cuneiform shape, and superficial situation which it generally assumes. In color, consistence, and friability, metastatic infarctions are entirely similar to those which arise from disease of the heart. The microscope also gives the same appearances.

When metastatic infarction terminates in metastatic pneumonia or abscess, discoloration and disintegration generally commence in the middle of the diseased part; cavities form, filled with a yellow mass, which consists of *débris* of the pulmonary substance, and of molecular decay of the extravasated blood and fibrin, but which at first does not contain any pus. Upon pouring water over its cut surface, we can see the vestiges of the lung floating in the hollow. The disintegration spreads gradually until scarcely a trace is left of former thickening, even at the periphery of the abscess. When situated immediately under the pleura, yellow croupous deposits form upon the latter, which cause the pleural surfaces to become adherent, and beneath it lies the infarction, "forming a rounded-nodular prominence like a furuncle" (*Rokitansky*).

SYMPTOMS AND COURSE.—We shall treat separately of the symptoms of hæmorrhagic infarction arising from diseased heart, and of those of metastatic infarction; since the appearance of the two forms of disease, in spite of their anatomical identity, varies in many respects on account of the difference in the diseases which cause them.

In many cases of chronic disease of the heart, hæmorrhagic infarction sets in with such well-marked and unequivocal symptoms, that its existence can be demonstrated with perfect certainty. In other cases the proof is difficult, or quite impossible.

The characteristic symptoms, from which we can infer the formation of one or more hæmorrhagic infarctions in a case of disease of the heart, are, a sudden dyspnœa, which may threaten suffocation, and a cough with a peculiar sputum tinged with blood. In many instances there are the signs of a circumscribed condensation of the lung, which are not unfrequently followed by those of pneumonia or of pleurisy. It is manifest that the stoppage of one or more branches of the pulmonary artery will produce extreme dyspnœa. As the process of respiration can only be carried on normally, when both the air in the vesicles and the blood in the capillaries are properly renewed, the arrest, either of access of blood, or of entrance of air into part of the lung, the obstruction, either of a bronchus, or of an arterial branch, must have an equal and extremely embarrassing effect upon respiration. The sputa, from the strong admixture of blood which they contain, bear a certain resemblance to pneumonic sputa, but they are less tough and almost always darker; and, moreover, the expectoration of this secretion is continued for a much longer time than is the expectoration of pneumonia. The former may persist for a week or even a fortnight. Circumscribed condensation of the pulmonary tissue can only be detected when the hæmorrhagic infarction is of comparatively large size, and has extended to the surface of the lung. The sound upon percussion then becomes dull, and crepitation and bronchial sounds are audible over a limited region of the chest. Although such cases occur, they are rare. The diagnosis may be confirmed, a few days after the attack of dyspnœa and bloody expectoration, by the development of extensive pneumonic infiltration, or of inflammatory effusions into the pleural sac, as we find that hæmorrhagic infarctions often produce inflammation of the surrounding pulmonary tissue, and still more frequently cause inflammation of the pleura.

Besides the symptoms hitherto described, and which are all immediately dependent upon stoppage of one or more branches of the pulmonary artery, there are, in many cases, other symptoms, which proceed from the thrombosis of the right heart, and hence are to be regarded as indirect tokens of hæmorrhagic infarction. These are, a sudden irregularity of the pulse, a sudden widening of the cardiac dulness, and the sudden cessation of an adventitious murmur, which had previously existed. This sudden subsidence of a loud, morbid sound is not only a most striking occurrence, but one which is generally very significant. My attention was first called to the full meaning of this symptom by the work of *Gerhardt* above alluded to; but I can fully confirm both the occurrence of the sign and its full diagnostic importance from my own experience. The picture of a hæmorrhagic infarction becomes very well marked, when the latter group of symptoms coexists with those described above. But emboli may break off, and be washed away from cardiac thromboses so small, that they produce no characteristic phenomena; hence, even where there are no signs of cardiac thrombosis, where the pulse remains regular, and where the cardiac dulness continues unchanged, we may still confidently diagnosticate hæmorrhagic infarction, whenever unequivocal signs of disorder of the circulation and capillary hæmorrhage of the lungs suddenly arise in the course of disease of the heart.

Finally, if we bear in mind that the characteristic expectoration of the blood from the air-vesicles is not always observed in hæmorrhagic infarction, and, moreover, that violent fits of dyspnœa may arise from a great variety of causes in disease of the heart, and that infarctions, seated deep within the lung, cause no physical signs, it is easy to understand why the disease, which, in many cases, does not present the smallest difficulty of diagnosis, may sometimes elude detection and even suspicion—as, for instance, where the patient is already extremely short of breath, and dropsical, and is otherwise wretchedly ill. In the dissection of cases of diseased heart, therefore, we should be prepared to find hæmorrhagic infarctions as "accidental discoveries" where their existence has not been suspected.

The liquid products of inflammation or of ulceration almost always pass into the circulation with the emboli; and, while the latter give rise to metastatic infarctions, the former result in the symptoms of pyæmia, septicæmia, intense fever, rigors, purulent inflammation of serous membranes, and the like.* We thus see why most patients with metastatic infarction of the lung are extremely depressed, why their sensorium is blunted by the intensity of the asthenic fever, and why they neither complain of pain in the side or breast, nor show any inclination to cough. In most cases there are neither subjective nor objective symptoms of disease of the lung. It is even the rule, at the autopsy of persons who have died of pyæmia and septicæmia during some suppurative or ulcerative process, to find metastatic infarction in the lungs, which, during life, was quite indistinguishable. These latent metastatic infarctions are easily accounted for, if we only call to mind the symptoms upon which diagnosis of the disease is based. The intense dyspnœa, which appears in cases where large arterial branches in the lung are obstructed, does not exist in metastatic infarction, where the occluded arteries are nearly always very small. Dyspnœa of slighter degree is not noticed by the patient in his stupefied condition. In like manner the characteristic sputum is almost always absent, as generally the patient neither coughs nor expectorates. Finally, notwithstanding their superficial position, metastatic infarctions scarcely ever occasion circumscribed dulness upon percussion, or produce bronchial breathing in the affected region. It is only in very rare cases that patients complain of piercing pain in some point of the chest, and expectorate thin, reddish-brown sputa. If, besides, a friction-sound be audible in the region of the pain, and if the original malady be one frequently productive of metastatic infarction in the lung—as, for instance, an injury of the skull affecting the diploë—we may pronounce our diagnosis with confidence; but, I repeat, that cases like this are very exceptional.

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* According to recent observations, the introduction into the blood not only of decomposed liquids, but even the absorption of liquid inflammatory products which are not decomposing, gives rise to violent fever, and to secondary inflammatory processes in distant parts of the body. It would thus seem as though pyæmia, which has been in some danger of disappearing from the list of diseases, may maintain its place by side of septicæmia.

TREATMENT.—The treatment of hæmorrhagic infarction can only be a treatment of symptoms. When the affection proceeds from disease of the heart, we must beware of attributing the dyspnœa to an aggravation of the pulmonary hyperæmia. We are aware that its real or chief cause is anæmia of portions of the lung. An injudicious venesection might have the effect of increasing a collapse of the lung already present, and of hastening a fatal issue. It is only when the obstruction of sundry arterial branches in the lung, has given rise to collateral hyperæmia, and to collateral œdema of the rest of the lung, and when the dyspnœa is plainly due in great measure to this cause, that cautious blood-letting, either by cupping or venesection, is ever admissible. As a general rule, until the pulse, which usually is feeble, grows stronger, and until the skin, which usually is cool, becomes warmer, we must confine our treatment to stimulation of the patient, and to the application of sinapisms and warm baths to the extremities. The expectoration of blood is rarely so abundant as to call for exhibition of the hæmostatic remedies recommended in a previous chapter. The inflammation of the lung or pleura, which often sets in at a later period, may demand local depletion, the application of cold and other antiphlogistic measures.

## CHAPTER VIII.

### PULMONARY HÆMORRHAGE WITH LACERATION OF THE PARENCHYMA.—APOPLEXY OF THE LUNG.

ETIOLOGY.—In this form of pulmonary hæmorrhage the tissues are destroyed by extravasated blood, and an abnormal cavity is established. Capillary hæmorrhage scarcely ever destroys the tissues of the lung. It is only erosion or laceration of the larger vessels, especially rupture of the arteries, which produces destruction of this kind. In rare cases atheromatous degeneration of the pulmonary artery causes its aneurismal dilatation and final rupture; but, more commonly, wounds, contusions, or concussions of the thorax, are the causes of pulmonary apoplexy.

ANATOMICAL APPEARANCES.—A cavity is found in the lung, containing both liquid and coagulated blood, and surrounded by tatters of the lacerated pulmonary substance. If the apoplexy have its seat on the periphery, the pleura, too, is often torn, and blood is poured into its sac. Such hæmorrhages are almost always fatal, so that we have little knowledge of the mode of repair of an apoplectic centre.

SYMPTOMS.—Violent and rapidly-fatal hæmoptysis, following serious injury of the thorax, or, in other cases, suffocation from effusion of blood into the bronchi, faster than it can be expectorated, or sudden death from internal hæmorrhage, may be the symptoms of this exceedingly rare disease, which, being absolutely deadly, is susceptible of no treatment.

## *INFLAMMATION OF THE LUNGS.*

Inflammation of the lungs may properly be regarded as of three kinds:

1. *Croupous pneumonia*, in which the air-cells are involved in a process identical with that which attacks the mucous membrane of the larynx in laryngeal croup.

2. *Catarrhal pneumonia*, a process intimately related to that already described as catarrhal bronchitis and laryngitis, producing an augmented secretion, and active generation of young cells (pus-corpuscles), but in which no *coagulable* exudation is formed. In both these varieties of inflammation the inflammatory products are thrown out upon a free surface, the tissue of the lung itself suffering no essential disturbance of nutrition.

3. *Interstitial pneumonia*, which is an inflammation involving the walls of the air-vesicles, and the interlobular connective tissue. As in the human subject, this latter form is always a chronic disease; it has been also called *chronic pneumonia*, in contradiction to the other varieties, whose course is usually acute.

## CHAPTER IX.

### CROUPOUS PNEUMONIA.

Etiology.—With regard to the pathogeny of croupous pneumonia, we refer to what has been said in the second chapter of the first section concerning croupous inflammation, and of its distinctness from diphtheria. In croupous pneumonia, also, a fibrinous, rapidly-coagulable exudation is thrown out upon the free surface of the air-vesicles, involving their epithelium, and including the newly-formed cells. Here, too, the vesicular walls become completely restored after expulsion of the exudation.

Sometimes pneumonia occurs under the influence of an acute dyscrasia, just as catarrh (as we have learned) attacks the air-passages in measles, exanthematic typhus, etc. This form of pneumonia, which accompanies typhus more often than it does other acute infectious disorders, may be distinguished by the name of *secondary pneumonia*, from the other varieties which arise more independently, and constitute a separate disease, which we may call *primary pneumonia*. It is wrong, however, to regard all cases of this disease, which supervene upon some chronic malady, as belonging to the secondary form.

The liability to primary croupous pneumonia exists at all periods of life, down to extreme old age. It is rare, however, among infants at the breast, and in the first years of childhood. Males are attacked more frequently than females; not, however, because vigorous, full-blooded persons are peculiarly subject to the disease. The latter, indeed, are by no means exempt; but feeble and broken-down subjects, convalescents from grave diseases, individuals who already have repeatedly suffered from pneumonia, are, perhaps, more liable to be attacked than the robust; and pneumonia often complicates diseases which have already effected an impoverishment of the blood, with emaciation and constitutional exhaustion. Very many of the inmates of hospitals, sufferers from inveterate disease, finally succumb to intercurrent pneumonia.

Its exciting causes are generally unknown. At times pneumonia becomes of very frequent occurrence, while croup, acute articular rheumatism, erysipelas, and other acute inflammatory disorders prevail at the same time, attacking their victims without any obvious provocative. This prevalence of acute inflammatory disease through the operation of unknown atmospheric and telluric agencies is generally spoken of as inflammatory epidemic influence. We particularly observe the epidemic occurrence of pneumonia in severe and protracted winters during the prevalence of a northeast wind. Sometimes, however, it arises under conditions precisely the reverse. The statistical statements as to the greater frequence of pneumonia in northerly and elevated localities have, of late, been regarded as untrustworthy.

Direct irritants acting upon the lungs, the inhalation of very cold or very hot air, foreign bodies, which have entered the air-passages and stopped up a bronchus, fractures of the ribs, wounds of the thorax, may be counted as among the exciting causes, although scarcely any of these conditions are found to exist in one case of pneumonia out of fifty. Nor is the croupous form of the disease often found to attack the parts about a morbid growth or around a hæmorrhagic infarction.

With regard to the influence of cold, it is difficult to decide in individual instances whether the attack has been preceded by an exposure to cold more severe than that to which the patient has repeatedly exposed himself with impunity. Opinions, therefore, are divided as to the effect of cold in producing pneumonia.

ANATOMICAL APPEARANCES.—Croupous pneumonia almost always attacks a somewhat extensive portion of the lung, commencing usually at the root, and spreading thence to the lower and afterward to the upper lobes. Sometimes an entire lung is inflamed, or the process may extend into the other lung, producing a double pneumonia. It is curious that in old persons and in cachectic individuals the mode of extension is usually different, as here the upper lobes are generally the first to be attacked, the lower not becoming involved until a later period of the disease. We distinguish three anatomical stages in pneumonia: 1st, the stage of engorgement with blood (*engouement*); 2d, the stage of hepatization; 3d, the stage of purulent infiltration.

In the first stage the pulmonary parenchyma is dark red, often reddish brown. It is heavier and firmer, has lost its elasticity, and pits upon pressure. Upon section, the inflamed portion of the lung does not crackle much, and a brownish or reddish liquid, of a strikingly viscid and tenacious nature, bathes the surface of the cut.

In the second stage, the air has disappeared from the air-vesicles, and the latter are filled by small, firm plugs of coagulated fibrin, to which an admixture of blood imparts a reddish color. A similar exudation has taken place in the extremities of the bronchi. The lung has now become remarkably heavy, sinks in water, does not crackle, is firm to the touch, but is very tender and friable. The appearance of its cut surface is granulated, especially when viewed by oblique light, and this is most distinct where the air-vesicles are large; less so in children, where they are small. The granules (which are merely the fibrinous plugs so often mentioned) can no longer be extracted from the lung by scraping with the scalpel, but adhere firmly to the walls of the air-cells. The granulated aspect of the cut-surface, the rigidity, the friability, the redness of the condensed lung, impart to it a considerable resemblance to liver, and thus the generally-adopted name of *red hepatization* has arisen. Sometimes, owing to spots of lighter color, and to deposits here and there of the black pigment which is secreted in the lung, together with the whiteness of the interior of the bisected bronchi and vessels, the section, instead of a uniform red, presents a variegated, "marbled," granite-like appearance. Afterward the redness fades more and more, either from cessation of the hyperæmia, or from disintegration of the hæmatin. The lung assumes a gray or yellowish appearance, while the texture continues in other respects the same, the pulmonary substance remaining rigid and granular (*yellow hepatization*). Besides the amorphous fibrin which fills the air-vesicles, the microscope reveals a very active formation of new cells, which probably spring from the epithelium of the vesicular walls. Should resolution set in, in the stage of hepatization, the fibrin and the young cells entangled in it undergo fatty metamorphosis and disintegration. An albuminous serum transudes from the walls of the vesicles; their contents become liquefied, converted into an emulsion, and finally are eliminated, partially by absorption, partially by expectoration. There is a slight deviation from the above when the pneumonic exudation is less fibrinous and less coagulable. The hepatized portion then is softer, its cut is smoother, and without distinct granulation. This is most common in the secondary pneumonia of typhus, and in that of old persons. When the pneumonia passes into the third stage, that of *purulent infiltration*, cell-formation assumes prominence, while the fibrin undergoes disintegration as in other cases. The granulated appearance is lost, the cut-surface is of a pale gray, or grayish yellow. A reddish-gray matter bathes its surface, and may be expressed in large quantities. The tissues are exceedingly tender, and are easily torn by the pressure of the finger. The minuter structure of the lung, however, is unaltered; the pulmonary tissue itself is still intact. Here, too, therefore, complete recovery may take place. The purulent contents may be ejected in part, and in part may undergo fatty degeneration and become absorbed.

The rarer sequelæ of pneumonia are.

1. *Formation of abscess.* The purely croupous form of inflammation with which we have here to do essentially excludes the idea of a destruction of the inflamed tissue. When abscesses form, the process has more of a diphtheritic nature. The proper tissue of the lung becomes infiltrated, and sloughs from the pressure of the fibrinous infiltration. In this way small cavities, filled with pus and *débris* of the pulmonary substance, form in the lung, which itself is infiltrated with pus. Sometimes they are solitary and sometimes they exist in great number. These collections of pus may increase in size from continual melting down of the tissues; several of them may coalesce, so that finally a huge abscess may occupy the greater part of the lung. These abscesses either end fatally through ulcerous phthisis, or else, in rarer instances, they open into the pleural sac. In other cases, a reactive interstitial pneumonia is set up in the parts adjacent, by which the abscess is incapsulated in a firm cicatricial tissue, its inner wall becoming smooth. Should a communication with the bronchi remain, its contents are evacuated from time to time, but are replaced by fresh matter generated by the interior surface. Should the cavity be closed, the pus may become thickened, and be converted into a cheesy paste, or, after disappearance of the organic substance, may change into a mortar-like or chalky concretion, which lies imbedded in an indurated firm scar.

2. *Gangrene of the lung* is a still rarer sequel to pneumonia. It appears only to occur when the supply of blood has been completely cut off from the inflamed portion of the lung by the formation of large coagula in the pulmonary arteries, and more especially when they form in the bronchial arteries, by whose means nutrition of the lung is carried on. The lung may become gangrenous even in the stage of red hepatization. The exudation then changes into a grayish ichorous liquid, and the pulmonary tissue breaks down into a blackish pulp. (See Chapter XII.)

3. The termination of croupous pneumonia in *cheesy infiltration* (or, as it is still too often called, *tuberculous infiltration*) is more common. If, in the second or third stage of the disease, when the fibrinous effusion and the cells which fill the vesicles take on fatty degeneration, the supply of serum effused by the walls of the vesicles prove insufficient, the fatty masses begin to dry up before their liquefaction is completed, and are converted into a more or less firm, yellow, cheesy substance. Hereafter we shall again refer to the subsequent changes occurring in this caseous infiltration of the lung, and shall express ourselves upon the inexpedience of permitting the imputation to arise of a sort of identity of the products of the latter process with those of tubercular granulation, by applying a similar title to both.

4. *Cirrhosis of the lung*, or *induration*, is finally to be mentioned as a rare termination of tedious pneumonia. This sequel is due to participation of the vesicular walls and the interstitial tissue in the process, when the disease is of long standing. Of this we shall treat more in detail in Chapter XI.

That portion of the lung which is not attacked by the inflammation is the seat of intense hyperæmia, as before stated; in fact, pulmonary œdema is, in many cases, the actual cause of death. Wherever the inflammation extends to the periphery of the lung the pleura also becomes implicated, showing minute arborescent injection and ecchymosis. It is then clouded and opaque, flabby, and covered with a thin layer of fibrin. Generally, the right side of the heart, from which the outflow of blood has been impeded by the stasis of the capillaries of the lungs, is overflowing with blood; the left heart, its supply being abnormally diminished, is less full. In like manner, and for the same reasons, stagnation of blood exists in the jugular veins, in the sinuses of the brain, and in the liver and kidney. The condition of the blood is exceedingly striking. The major part of that which is in the great vessels is not liquid, but is coagulated into firm yellow masses. Lumps of curdled fibrin exist in the heart, where they are firmly entangled amid the trabeculæ and under the valves; and long, firm, tough, polypous coagula may be drawn out of all the arteries.

Symptoms and Course.—We shall discuss the subject of secondary pneumonia in treating of typhus, etc., as it is impossible to draw up a picture of this disorder without making a detailed analysis of the symptoms of the disease upon which it depends.

The commencement of primary pneumonia, in almost all cases, is announced by a rigor which may last for half an hour, or even for several hours, before giving place to a sensation of heat. As is well known, the cold is a mere subjective symptom, and the temperature is appreciably elevated, even during the algid stage.

This rigor is important both in a diagnostic and in a prognostic point of view. In no other affections, excepting intermittent fever and septicæmia, do we encounter chills of equal violence; and in the latter disorders the paroxysms are repeated, while the rigor which ushers in pneumonia is almost always the only one throughout the entire course of the illness. It is from this chill that we calculate, in counting the duration of the disease. In children, convulsions often occur instead of a chill.

The elevation of temperature, which rises to 103° or 105° Fahrenheit (rarely higher), even on the first day, is accompanied by acceleration of the pulse, and by increase of thirst. The countenance is red; the patient complains of pain in the back and loins, and of a distressing soreness of the limbs. There is great prostration and muscular debility. The tongue is coated, and the appetite entirely gone; occasionally there is vomiting. As these symptoms often precede the local manifestations by one and even two days, they used formerly to be attributed by many to the accumulation of fibrin in the blood (hyperinosis). Some have even gone so far as to ascribe a critical significance to pneumonia (*Dietl*), and to declare that the disorder only disappeared after the elimination of the superfluous fibrin from the blood. All these symptoms, however, appertain to the fever, and are more or less pronounced in all febrile diseases, whether the fibrin of the blood be increased or diminished in quantity, or whether its quantity remain unchanged. We need not demonstrate more fully, that every fever, by increasing the rate of transformation and consumption of the tissues, must thereby alter the composition of the blood, and that the products of the interchange of materials are mingled with the blood in greater quantity. This febrile crasis and the elevated temperature of the blood sufficiently account for the perversions of nutrition and function, which take place in febrile affections—constitutional disturbance of fever.

Although fever and derangement of the general health are of earlier occurrence than the symptoms of nutritive derangement which the lung has suffered, yet we may often observe the same thing in febrile catarrhs and other inflammatory fevers. We may assume in such cases that the inflammatory disturbances of nutrition commence quite as soon, at least, as the fever, but that for a while they do not betray themselves by causing pain, cough, or dyspnœa, but remain latent. In other instances, symptoms of functional disturbance appear in the lung either simultaneously with the chill or immediately afterward.

The first of these is shortness of breath, a constant accompaniment of pneumonia. Assuming the normal rate of breathing of adults to be twelve, sixteen, or twenty breaths a minute, we see it augmented in pneumonia to forty and even fifty breaths, and find it to attain a still come incapsulated, and its contents may thicken into a cheesy or calcareous mass.

Gangrene of the lung, as a result of hæmorrhagic infarction, will be described in Chapter XII.

In explaining the pathogeny of metastatic infarction, we have already alluded to the small volume, the cuneiform shape, and superficial situation which it generally assumes. In color, consistence, and friability, metastatic infarctions are entirely similar to those which arise from disease of the heart. The microscope also gives the same appearances.

When metastatic infarction terminates in metastatic pneumonia or abscess, discoloration and disintegration generally commence in the middle of the diseased part; cavities form, filled with a yellow mass, which consists of *débris* of the pulmonary substance, and of molecular decay of the extravasated blood and fibrin, but which at first does not contain any pus. Upon pouring water over its cut surface, we can see the vestiges of the lung floating in the hollow. The disintegration spreads gradually until scarcely a trace is left of former thickening, even at the periphery of the abscess. When situated immediately under the pleura, yellow croupous deposits form upon the latter, which cause the pleural surfaces to become adherent, and beneath it lies the infarction, "forming a rounded-nodular prominence like a furuncle" (*Rokitansky*).

SYMPTOMS AND COURSE.—We shall treat separately of the symptoms of hæmorrhagic infarction arising from diseased heart, and of those of metastatic infarction; since the appearance of the two forms of disease, in spite of their anatomical identity, varies in many respects on account of the difference in the diseases which cause them.

In many cases of chronic disease of the heart, hæmorrhagic infarction sets in with such well-marked and unequivocal symptoms, that its existence can be demonstrated with perfect certainty. In other cases the proof is difficult, or quite impossible.

The characteristic symptoms, from which we can infer the formation of one or more hæmorrhagic infarctions in a case of disease of the heart, are, a sudden dyspnœa, which may threaten suffocation, and a cough with a peculiar sputum tinged with blood. In many instances there are the signs of a circumscribed condensation of the lung, which are not unfrequently followed by those of pneumonia or of pleurisy. It is manifest that the stoppage of one or more branches of the pulmonary artery will produce extreme dyspnœa. As the process of respiration can only be carried on normally, when both the air in the vesicles and the blood in the capillaries are properly renewed, the arrest, either of access of blood, or of entrance of air into part of the lung, the obstruction, either of a bronchus, or of an arterial branch, must have an equal and extremely embarrassing effect upon respiration. The sputa, from the strong admixture of blood which they contain, bear a certain resemblance to pneumonic sputa, but they are less tough and almost always darker; and, moreover, the expectoration of this secretion is continued for a much longer time than is the expectoration of pneumonia. The former may persist for a week or even a fortnight. Circumscribed condensation of the pulmonary tissue can only be detected when the hæmorrhagic infarction is of comparatively large size, and has extended to the surface of the lung. The sound upon percussion then becomes dull, and crepitation and bronchial sounds are audible over a limited region of the chest. Although such cases occur, they are rare. The diagnosis may be confirmed, a few days after the attack of dyspnœa and bloody expectoration, by the development of extensive pneumonic infiltration, or of inflammatory effusions into the pleural sac, as we find that hæmorrhagic infarctions often produce inflammation of the surrounding pulmonary tissue, and still more frequently cause inflammation of the pleura.

Besides the symptoms hitherto described, and which are all immediately dependent upon stoppage of one or more branches of the pulmonary artery, there are, in many cases, other symptoms, which proceed from the thrombosis of the right heart, and hence are to be regarded as indirect tokens of hæmorrhagic infarction. These are, a sudden irregularity of the pulse, a sudden widening of the cardiac dulness, and the sudden cessation of an adventitious murmur, which had previously existed. This sudden subsidence of a loud, morbid sound is not only a most striking occurrence, but one which is generally very significant. My attention was first called to the full meaning of this symptom by the work of *Gerhardt* above alluded to; but I can fully confirm both the occurrence of the sign and its full diagnostic importance from my own experience. The picture of a hæmorrhagic infarction becomes very well marked, when the latter group of symptoms coexists with those described above. But emboli may break off, and be washed away from cardiac thromboses so small, that they produce no characteristic phenomena; hence, even where there are no signs of cardiac thrombosis, where the pulse remains regular, and where the cardiac dulness continues unchanged, we may still confidently diagnosticate hæmorrhagic infarction, whenever unequivocal signs of disorder of the circulation and capillary hæmorrhage of the lungs suddenly arise in the course of disease of the heart.

Finally, if we bear in mind that the characteristic expectoration of the blood from the air-vesicles is not always observed in hæmorrhagic infarction, and, moreover, that violent fits of dyspnœa may arise from a great variety of causes in disease of the heart, and that infarctions, seated deep within the lung, cause no physical signs, it is easy to under-sides, the temperature sometimes reaches a height greater than any previously attained.

The pulse, whose frequence in a pneumonia of average severity usually ranges between ninety and a hundred and twenty beats a minute, may in severe cases, where the temperature is very high, attain a frequence of a hundred and thirty, or a hundred and fifty or more. While, at the outset of the attack, it usually is large and full, as the malady progresses, it often becomes small and soft. In some cases, this is due to depression of the heart's action, by the high temperature (which always tends to produce asthenia), so that its feeble strokes scarcely overcome the resistance opposed by the aorta to the outflow of the blood. Under these circumstances (upon the principle that the effect is in proportion to the power, and in inverse proportion to the resistance), but little blood is expelled from the heart, causing a feeble pulse-wave, and a small pulse. In other, and probably in the majority of cases, it is not the weakness of the cardiac contractions, but the lack of blood in the left ventricle, which causes a deficit in the supply of the aortic system, and renders the pulse small and soft. The left ventricle is imperfectly filled, because afflux of blood to it is obstructed. In an extensive pneumonia, an obstacle to the circulation arises (partly from the inflammatory stasis, partly owing to pressure of the exudation upon the capillaries), which cannot be fully compensated for by acceleration of the capillary circulation in the uninflamed portion of the lung. The consequence is, that too little blood enters the left heart, while the right heart and the veins of the aortic system are overloaded. (Upon cutting into a piece of hepatized lung, but little blood flows from it. The redness in the beginning of hepatization depends upon extravasation. The lack of blood of the inflamed part is most conspicuous in yellow and gray hepatization, and in purulent infiltration.)

The blueness of the lips and cheeks, which is observed in severe pneumonia, is also dependent upon the disturbance of the pulmonary circulation, and upon impediment to the outflow of blood from the right ventricle, and from the veins of the aortic circulation; but we have no acceptable explanation of the reddening of the cheek, which often occurs at the side upon which the pneumonia exists. In many instances a herpetic eruption develops upon the second or third day upon the lips, more rarely upon the nose, cheeks, or eyelids; and from the frequence of herpes during pneumonia, and its great rarity in abdominal typhus, and other diseases, the appearance of vesicles filled with a clear liquid may be of diagnostic value in doubtful cases.

The headache, by which the invasion of pneumonia is accompanied, usually continues throughout the attack. It is generally combined with sleeplessness, or the sleep is troubled by dreams; and, if the patient be at all of an irritable temperament, there is apt to be slight delirium. These symptoms are mainly due to the fever, and cease as soon as the fever subsides. We must beware of inferring the existence of grave cerebral disease from the presence of these signs alone.

Even where there is no complicating gastric disorder, the appetite usually is lost, the tongue is lightly coated with white, and shows a tendency to dryness, the thirst is considerably augmented, and the stools are dry and constipated. These symptoms are also the result of fever, and occur in almost every other febrile complaint. The loss of appetite is the most difficult to account for. One would suppose, *a priori*, that the augmented destructive assimilation which takes place during fever, by means of which the high temperature of the body is maintained, would occasion an increased demand on the part of the system for a compensating supply of nourishment to replace the waste, and we are quite at a loss to understand why no such want is usually felt by the patient. The coated tongue, its tendency to dryness, as well as the aggravated thirst (see catarrh of the oral mucous membrane), and the dryness of the stools, are satisfactorily accounted for by the increased evaporation of liquid from the skin, in consequence of which the tissues become dryer and their secretion is diminished.

Obstruction to the flow of blood from the liver not unfrequently leads to a perceptible enlargement of that organ. Perhaps, in some cases, the slight jaundice which occurs during pneumonia is dependent upon this obstruction of circulation in the liver, and is analogous to the icterus which appears, from the same cause, with tolerable frequence in disease of the heart. As the hepatic veins are intertwined with the biliary ducts, distention of the former may result in compression of the latter, and thus cause retention and absorption of bile. However, this theory of the origin of icterus is only to be admitted when the liver is greatly swelled and the patient is extremely cyanotic. Far more frequently, the symptoms of jaundice during pneumonia depend upon a catarrh of the duodenum and of the biliary ducts, or else it arises from "dissolution" of the blood—that is to say, a disintegration of the blood-corpuscles, by which free coloring-matter of the blood is converted into biliary coloring-matter outside of the liver.

The pneumonic process and the fever which attends it exercise an important influence upon the constitution of the urine. While the fever lasts the proportion of water in the urine is reduced by the insensible perspiration. The urine is scanty and concentrated, its color is somewhat dark, and its specific gravity is high.

Among the solid constituents of the urine, the urea is considerably increased in quantity. As is well known, the ultimate products of destructive assimilation of nitrogenous tissues are eliminated under the form of urea and uric acid. The elevation of the temperature of the body in febrile disease depends upon an abnormal generation of heat from a morbidly active combustion of the constituents of the tissues, in which, of course, the nitrogenous elements participate. A short fever reduces the weight of the patient far more than does a fast, without fever, of much longer duration. But the patient not only grows thin because his fat is consumed in overheating his body, but the muscles undergo a marked atrophy, and a considerable period of time elapses ere a convalescent from fever regains his former strength, and ere his muscles are restored to their original volume. The increased destructive assimilation of the nitrogenous constituents of the body during fever is also susceptible of direct proof, by the absolute or relative augmentation in the production of urea. Patients suffering from pneumonia, with violent fever, even though their diet be absolutely non-nitrogenous, eliminate quite as much urea in their urine, if not more, than a healthy person does whose food consists almost entirely of meat and eggs. I have known pneumonia patients to excrete forty grammes of urea within twenty-four hours, while one of my pupils, who was in good health, and whose diet was precisely that of the sick man, passed but from thirteen to fifteen grammes in the same time. The urine very commonly becomes turbid upon cooling, from precipitation of its urates; but it appears to me that this phenomenon is due rather to the reduced proportion of water in the urine, which thus becomes incapable of holding the urates in solution at a low temperature, than to an excessive formation of the salts themselves. By gently warming the urine the urates can always be redissolved, and the cloudiness of the urine be dissipated.

While the urea of the urine is increased in quantity, the amount of inorganic salts which it contains, especially its alkaline chlorides, is diminished, and at the height of the disease they may disappear completely. If we add a few drops of a solution of nitrate of silver to some of the urine, previously acidulated, the precipitate of chloride of silver, so distinct in healthy urine, is scarcely, if at all, observable. The greater part of this chloride of sodium depends, no doubt, upon the use of food containing salt, and the diet of a pneumonia patient might account for the diminution of alkaline chlorides in the secretion; but as, even in starving animals, small quantities of alkaline chlorides are found in the urine, as a product of transmutation of their tissues, its complete disappearance in pneumonia cannot be attributed solely to the diet of the patient: and we are warranted in supposing that the portion of alkaline chlorides produced by destructive assimilation is excreted from the blood with the pneumonic exudation.

The concentration of the urine, the augmentation of urea, the diminution of the chlorides, as well as the appearance of biliary pigment in the urine, are simply due to the improper quality of the matter conveyed to the kidneys for the production of urea. The appearance of albumen in the urine, which not unfrequently occurs in severe pneumonia, is dependent upon other causes. Its presence is sometimes occasioned by engorgement of the emulgent veins. As is well known, albuminuria may be produced artificially in the lower animals by ligation of these veins. The chief source of the albuminuria of heart-disease is obstruction of the venous circulation of the kidney. The presence of albumen in the urine of pneumonia, however, is only to be ascribed to such a cause when it is accompanied by cyanosis, enlargement of the liver, and other evidence of intense venous engorgement of the greater circulation. In most cases its source is in the parenchymatous degeneration of the kidneys, of which we shall speak more fully in our second volume, and which consists in a swelling and opacity and molecular destruction of the renal epithelium. This parenchymatous degeneration of the kidney, with its consequent albuminuria, occurs in a great variety of febrile disorders, and is apparently a result of excessive elevation of the temperature of the body, or febrile crasis. The more intense the fever, so much the more probably will albumen be found in the urine of pneumonia patients, although there may be scarcely any sign of venous engorgement of the systemic circulation. The skin, which, at the commencement of the attack, usually is dry and parched, after a day or two often becomes moist, and even bathed in sweat, without, however, affording any material relief to the patient.

Hitherto we have been describing the *stadium incrementi*, the forming stage of the disease. Its transition into the *stadium decrementi*, or stage of decline, is not gradual, but takes place with a suddenness without parallel in any other disorder.

In former editions of my book I have asserted with great positiveness that the crisis of a pneumonia almost constantly arrived either on the fifth, seventh, or, in rare instances, upon the third day, and I believed this assertion to be warranted by the results of a large number of observations. Meanwhile I have become satisfied that the ancient doctrine, that the crisis of pneumonia always occurred on the odd days, is untenable, in spite of the high modern authorities who have pronounced in favor of its correctness. If, in calculating the duration of the disease, we take accurate notice of the hour at which the initiatory chill began, and of that in which the decline of the fever commenced, it will be seen that the critical period of pneumonia takes place quite as often upon the even days as upon the odd ones. For instance, a pneumonia, which begins by a chill on Monday at noon, culminates, no doubt, in many cases, in course of the following Sunday; but the crisis occurs quite as often during the forenoon (hence during the sixth day) as in the afternoon or seventh day. (As I hear from one of my pupils, in a certain great university city, which is fond of being called the metropolis of intelligence, all the pneumonias at one clinique terminate on the odd days, and at another on the even ones.)

The symptoms continue with constant or increasing intensity until the critical day, which generally arrives toward the end, less commonly about the middle, of the first week of the disease; and while the condition of the patient, from the dyspnœa, the thirst, and the intense constitutional disorder, is beginning to awaken an earnest solicitude, a striking change takes place, often within a few hours. The temperature and the frequence of the pulse often sink rapidly, the dyspnœa abates, the patient feels easier and more free. In course of twenty-four hours convalescence is often fully established. The patient sleeps, calls for food, and merely complains of extreme debility. From this time the recovery of many patients progresses steadily. The temperature not unfrequently falls below the normal standard, and I repeatedly have seen the pulse sink to forty beats a minute, although the patient had not taken a grain of digitalis. The blood disappears from the expectoration, sometimes gradually, sometimes with suddenness. The sputa become somewhat more copious, but generally to so slight a degree that we are compelled to suppose that the greater parts of the exudation must be absorbed, and that but little of it is expectorated. The tenacity and transparence of the sputum disappears with the blood; it becomes yellowish—sputa cocta. The yellowness depends upon an admixture of young cells, which show more or less trace of fatty metamorphosis. Besides slightly granular pus-corpuscles, cells filled with oil-globules, fat granule-cells, and collections of granules, and of free oil-molecules, and black pigment-cells in greater or less numbers, are found in the expectoration. Although reabsorption commences very soon after exudation is complete, yet a considerable period of time usually elapses before auscultation and percussion show that the pneumonic infiltration has completely disappeared.

In subjects previously healthy, the course of the vast majority of pneumonias is as above described. Indeed, with the exception of the infectious diseases, there are few maladies whose average course is so remarkably uniform. That we should not, until recently, have perceived its evidently cyclical character, is owing to the active manner in which we used formerly to attack the disease whereby its typical course became deranged. One must bear in mind that not very long ago it would have been thought a crime to treat a pneumonia without blood-letting, and even without repeated venesection.

In some cases the crisis does not occur at the end of the first week, or there is but a short remission, after which the disease grows worse again, and continues into the second week. The pneumonic infiltration continues to spread, the temperature remains high, and is sometimes higher than ever. Signs of extreme prostration now set it, due in part to the elevation of the temperature, partially also to exhaustion produced by continuous and excessive calorification, and to the profuseness of the exudation, which I have repeatedly estimated at three pounds after comparison of the weights of the diseased and healthy lung. The fever, formerly of "inflammatory" type, now assumes an asthenic, "nervous" (typhoid) character. The pulse grows extremely frequent, small, and soft; the tongue becomes dry and incrusted; all the senses are blunted; the patient not unfrequently voids his urine and fæces involuntarily, in the bed; some patients lie in a stupor from which they can scarcely be roused; others, again, are wildly delirious, so that it is scarcely possible to restrain them in bed. In many such cases, especially if the patient have not been depleted by blood-letting, a change for the better may still take place toward the end of the second week, and again the transition from a condition apparently desperate, to one of almost complete convalescence, may then occur in the course of a few hours.

The crisis at the end of the first week fails to occur also, when the stage of hepatization passes on into that of purulent infiltration, and the fever continues into the second week with equal or even aggravated intensity. Here, too, the pulse is usually small, and the mouth is dry and sticky. The patients are somnolent, or else delirious; the temperature, especially in the evening, is greatly elevated, and sometimes there are slight chills. The sputa, which are generally profuse, contain great quantities of cells in a state of fatty degeneration. It is clear that auscultation and percussion alone can distinguish an extension of the process of hepatization from the transition into purulent infiltration.

When the pneumonia attacks aged persons, or subjects of depraved constitution, adynamic symptoms may arise, even though the malady be not of unusual duration and although purulent infiltration have not occurred. Indeed, so promptly do they sometimes develop, so immediately do they appear after the chill, and the first onset of the fever, that the signs of pulmonary disorder are entirely eclipsed by those of grave asthenic fever. As we have stated already, many patients of this kind have no cough and *no characteristic sputa; nor do they complain either of dyspnœa or of pain.* The frequency of respiration is often ascribed to the fever, and patients sometimes die with the diagnosis of a "typhoid influenza," a catarrhal fever, or a "typhoid gastric fever," whose autopsy reveals extensive pneumonic infiltration; the physician having been deceived by external appearances, which really bear greater resemblance to typhus than to pneumonia of vigorous adults, and having neglected to make a physical exploration of the chest.

Asthenic fever may also develop, sometimes, in subjects previously healthy and vigorous, where *pneumonia* is *complicated with acute gastric or intestinal catarrh.* True, such cases, which are not rare, differ from the pneumonia of old persons, inasmuch as the pain, cough, and characteristic sputa are not at first absent; but the depressing effect of the complication as well as the fever, which is usually of great intensity, soon result in an extreme prostration and in other symptoms, which create the terrifying impression upon the minds of the laity that the malady has become "typhoid" ("nervous"), or "that a nervous fever has set in." The disease is further disguised, and the diagnosis rendered doubly obscure, by the thickly-coated tonge, which afterward often becomes incrusted with black scabs, by the distended abdomen and by the watery discharges from the bowels, and—if the intestinal catarrh have also involved the ductus choledochus—by the jaundiced hue of the skin, and sclerotica. Here, too, physical examination is our sole safeguard against error and mortifying *post-mortem* disclosures. Pneumonia is apt to assume very peculiar characteristics when it attacks persons of intemperate habits. The beginning of the attack seems rather to be a fit of delirium tremens, and the symptoms of perverted cerebral action are so prominent that the pulmonary affection is liable to escape notice. The patient can hardly be kept in bed; he is exceedingly loquacious, does not complain, but declares that he is perfectly well. He is in a most cheerful humor, and his delirium and illusions are of that peculiar kind which is almost pathognomonic of delirium tremens. He sees small animals, especially mice and beetles, picks with great industry and persistance at his bed-clothes, or executes all the manipulations of his avocation in pantomime. Even though a patient in this condition have no cough, no expectoration, and complain of no pain, yet his chest should be explored with great care, especially if he have fever. Many a patient has died in a strait-jacket with a diagnosis of delirium tremens, whose real disease has been pneumonia. At a later period the scene changes. It is a well-known fact that drinkers are equally incapable, or even still less capable, of bearing an increase of calorification and an augmentation of their animal heat than aged or debilitated persons, and that a fever of very moderate intensity and brief duration exercises an exceedingly depressing and exhausting influence upon the vigor of the heart, the action of the brain, and upon all other functions. In a very few days the pulse, originally full, grows small and weak, the extreme excitement and bustling demeanor give place to a deep apathy, and to rapidly-increasing somnolence, the skin is bathed in sweat (from incipient palsy of the cutaneous muscles), gurgling sounds arise in the chest (from commencing paralysis of the muscles of the bronchi)—and the patient dies with the symptoms of œdema of the lung.

With regard to the termination of pneumonia, we have already seen that recovery is often rapid, where the exudation liquefies, and is reabsorbed after completion of the stage of hepatization. Complete restoration may also take place from the stage of purulent infiltration, only, as the patients are exhausted by weeks of fever, their convalescence is extremely tedious.

Death, during the first and second stages of pneumonia, usually proceeds from hyperæmia and collateral œdema, by which the uninflamed air-vesicles are rendered incapable of carrying on respiration. Much more rarely it depends upon the excessive extension of the pneumonic infiltration alone. The intense dyspnœa, profuse frothy or liquid sputa, moist *râles* in the uninflamed parts of the lung, the sudden sinking of the patient, the drowsiness, the vomiting, the coolness of the skin, are all signs of insufficient respiration, and of imminent danger of carbonic-acid poisoning. Unless aid be at hand, the symptoms of palsy soon prevail, and the patient succumbs under symptoms of œdema of the lung, palsy of the bronchi, and of suffocative effusion.

A fatal issue, during the stage of red hepatization, resulting from engorgement of the cerebral veins, with consequent effusion, is of far rarer occurrence. Simple blueness of the face need not lead us to fear cerebral congestion; nor are even the headache and the delirium sufficient to warrant such apprehension, neither do they require the active treatment imperatively demanded by that condition. If, however, the patient fall into somnolence which cannot be ascribed to the embarrassment of respiration, or if he complain of a sense of formication, or of numbness of his limbs, or should slight convulsions occur, life is undoubtedly threatened by œdema of the brain, and death may ensue with the symptoms of coma.

The third and most usual cause of death, during the stage of red hepatization in pneumonia, is exhaustion. From this cause a comparatively slight attack of this disorder is extremely dangerous where the patient is old or debilitated, or where he is a drunkard whose nerves are in constant need of stimulus, who trembles until he has his dram, and in whom the privation of the supply, added to the prostration produced by the fever, soon brings on paralysis. In like manner, a complication with intestinal catarrh and icterus tends to hasten the exhaustion; or, finally, the longer duration of the fever, and the magnitude of the exudation in a protracted pneumonia, may expend the strength of a person previously vigorous and healthy. In all these cases the obtuseness of the sensorium increases to stupor, the pulse becomes smaller and smaller, the skin is bedewed with clammy sweat, and the patient dies from passive hyperæmia, passive œdema, and suffocative effusion.

Death takes place with symptoms quite like these in the third stage, that of purulent infiltration, when the strength proves insufficient to withstand the duration and intensity of the fever. Sometimes the asthenic symptoms, which may arise during pneumonia, are accompanied by another group of symptoms of a different kind. The pulse grows small and irregular, a slight jaundice appears, which manifestly does not depend upon biliary obstruction; the urine becomes albuminous, the mind of the patient is much disturbed, the delirium being violent at first, afterward settling into stupor. When there is much jaundice, this description corresponds nearly with that of the *bilious pneumonia* found in many of the ancient pathologies. In these cases we probably have to do with a parenchymatous degeneration of the heart, liver, kidneys, brain, and blood. In their appropriate sections we shall consider in detail the subject of parenchymatous degeneration of these organs, as well as the relation of icterus to parenchymatous degeneration of the liver, and then dependence of this degeneration upon an increase of the animal heat, and upon intensity of the febrile crasis.

With regard to the rarer sequelæ of pneumonia, we may have good reason to suspect the formation of an abscess, when the slight shivering fits, which accompany purulent infiltration, change into violent rigors; and when a yellow-gray discharge, containing more or less pigment, begins to be expectorated in large quantities; but the diagnosis is only sure, when, by means of the microscope, we can discover elastic fibres, which, from their structure, are recognizable as belonging to the lung, or when physical exploration shows the existence of a large cavity in the chest. When a pulmonary abscess ends fatally, death takes place under conditions similar to those which accompany death from purulent infiltration. If the abscess heal, the expectoration loses its yellow color, little by little, as the cavity gradually becomes enveloped in a capsule of connective tissue; and when the abscess is completely closed the sputa cease entirely. Should a permanent cavity remain behind, lined with a pyogenic membrane, and surrounded by indurated connective tissue, it affords the same symptoms, runs the same course, and gives rise to the same danger as do the bronchiectatic cavities of which we have to treat in Chapter XI. The formation of new connective tissue and its contraction in the regions about the cavity also give rise to those depressions of the thorax which we shall describe by-and-by. Gangrene—a very rare sequel to pneumonia—is characterized by most intense collapse, by the expectoration of a blackish sputum of a most foul, putrid odor, together with the physical signs of a cavity in the lungs (see Chapter XII.).

Caseous infiltration, as a sequel to pneumonia, is by no means confined to patients in whose lungs old deposits of tubercle already exist, but may also take place in subjects previously in good health. Especially is this the case with emphysematous persons when attacked by croupous pneumonia of the lungs, which is rare. In such cases, although the fever moderates somewhat upon the critical day, it does not subside so completely as when it terminates in resolution. The patients do not improve, the cough and dyspnœa remain. In the evening the pulse is more frequent; auscultation and percussion reveal a persistent condensation of the parenchyma of the lung. After some time, the infiltration dissolves, causing vast destruction of the lung, the symptoms of which we shall examine more closely when considering the subject of pulmonary consumption.

For the termination of croupous pneumonia in induration or cirrhosis of the lung, see Chapter XII.

*Physical signs of Croupous Pneumonia.—Inspection* gives negative results as regards the contour of the thorax. Both sides of the chest preserve their normal dimensions, and the intercostal spaces present their proper shape of shallow furrows, a condition of great importance in distinguishing pneumonia from pleuritis. There is, however, a decided modification of the respiratory movements, since at the beginning of the attack the patient favors the affected side on account of the pain; and, as in the later stages of the disease, the vesicles are filled with exudation, and hence are impervious to the air. It is often possible to recognize the side upon which the pneumonia has its seat at the first glance, as the healthy side heaves normally, while the inflamed side, as it were, lags behind. When both lower lobes are infiltrated, the diaphragm cannot descend; and the epigastrium does not project upon inspiration. The patient breathes by dilatation of the upper part of the chest alone (costal type).

The first point which strikes the attention, upon *palpation*, is an intensification of the impulse of the heart, and (what is very important in distinguishing between pleuritis and pneumonia) the heart-shock is felt in its normal situation. Palpation also reveals that, during the period of engorgement, and often during that of hepatization, the vibrations of the chest are unusually distinct and strong, when the patient speaks—*that the pectoral fremitus is strengthened.* This important diagnostic sign may grossly mislead any one, who is ignorant of the fact that, in almost all healthy persons, the pectoral fremitus is stronger upon the right side than upon the left. This is probably due to the circumstance that the right bronchus is wider, shorter, and stands almost at a right angle with the trachea, while the left is longer, and narrower, and passes off from the trachea more obliquely. (*Seitz.*)

The morbid intensity of the pectoral fremitus, during the stage of engorgement, depends upon the loss of elasticity which the pulmonary tissue sustains at this period. Under normal conditions, the transmission of the vibrations from the trachea and larger bronchi to the thoracic wall is impeded by the tension of the elastic vesicles; moreover, the elasticity of the healthy lung exerts a sort of suction upon the inner surface of the thorax, whereby thoracic vibrations are held in check. These two forces, by which the normal vocal resonance is enfeebled during health, are removed when the elasticity of the lung is destroyed. The still further increase of the vocal fremitus, which is often observed during the stage of hepatization, is owing not only to the loss of elasticity of the hepatized lung, but also to the fact that the vibrations which the vocal chords have imparted to the air within the trachea and bronchi pass unimpaired to the walls of the chest, as the medium through which they are transmitted is no longer an interrupted one (alternations of air and vesicular wall), but a continuous one, the solidified pulmonary parenchyma. It sometimes happens that the transmission of the vibratile waves is checked by a temporary occlusion of the bronchi by secretion; but we not unfrequently observe instances, in which the pectoral fremitus over a hepatized point is permanently weakened or is entirely deadened, when there is neither bronchial obstruction nor pleuritic exudation. In such cases we may infer that the close contact of a compactly-infiltrated lung prevents the walls of the chest from vibrating.

*Percussion* during the stage of engorgement often gives rise to a purely tympanitic, hollow sound. The elasticity of the normal lung may be compared to that of a tightly-inflated bladder; its ring is not tympanitic. In the stage of hepatization the vesicles having lost their elasticity, its condition is like that of a cluster of imperfectly-inflated bladders. Its percussion-sound then is tympanitic. The "*hollow*" percussion-sound depends upon a diminution which the exudation causes in the amount of air contained in the vesicles, thereby reducing the size of the vibrating body. We regard the expressions "*full*" and "*hollow*" as thoroughly intelligible and practical. By universal custom, the sound produced by the vibration of a large voluminous body is called a "full" tone, and that proceeding from the vibration of a small body is called a "hollow" tone. Thus, the percussion-sound of the stomach sounds full to the ear of the beginner; that of the small intestine hollow. I find that there are few practitioners who can recognize with facility that the tympanitic percussion-sound of engorgement also is hollow, while for many it is difficult to make out its higher pitch.

During hepatization, when the solidified point lies in immediate contact with the side of the chest—but only in such a case—the percussion-sound is deadened, and during the act of percussion an increase of resistance is felt over the point struck. This is because a hepatized lung, like any other compact body void of air, cannot be made to vibrate. The thicker and wider the hepatized region lying in contact with the chest, so much the more marked are the dulness and resistance. When the dulness is but slight, it will generally be perceived that the sound is also hollow; when the sound is perfectly dull, the full and hollow tones cannot be appreciated. When the seat of the disease is central, that is to say, at the roots of the lung, very extensive hepatization of the lung may exist without alteration of the sound upon percussion.

Auscultation during the stage of engorgement usually affords a crackling sound to the ear, like that which is heard when one throws salt into the fire, or when a few hairs are rubbed between the fingers before the ear. This crackling (*Laennec's râle* crepitant), which is formed in the minute spaces of the bronchial terminations and pulmonary vesicles, is the finest of all the moist *râles*, and, as the fluid in which it arises is extremely viscid, it is also the dryest of the moist *râles*. Their mode of origin perhaps is, that the vesicular walls, which during expiration became glued together, are forcibly separated by the air which enters upon inspiration. As soon as that portion of the lung which touches the thoracic wall is completely infiltrated, vesicular breathing is arrested, as the vesicles there are impenetrable to the air. Instead *bronchial respiration* is heard, that is to say, we hear the sound which the to-and-fro movement of the air in the trachea and larger bronchi is always making, but which is not transmitted to the ear through the healthy lung, the structure of which, consisting of alternations of air and vesicular wall, furnishes a poor conducting medium. When, instead of this bad conductor of sound, a uniform medium lies between the ear and the bronchi, these bronchial sounds become audible; always provided, that the bronchi communicate with the trachea, so that the air may either pass to and fro in them, or that the air which they already contain may be set in vibration with every breath. Moreover, the bronchi in the condensed part of the lung form better conductors of sound than those which traverse the normal lung. If the bronchi should be filled up by accumulated secretion, as often happens temporarily in the third stage of pneumonia, the bronchial breathing ceases, and does not again become audible until the bronchi have become emptied by coughing.

Bronchophony arises under conditions similar to those under which bronchial respiration is produced. The vibrations of the vocal chords during speech are conducted along the column of air in the larger bronchi, but are only perceptible upon the surface of the chest as an indistinct buzzing, as long as the healthy pulmonary substance lies between the ear and the bronchi; the healthy lung-substance being, as we know, a bad conductor of sound. If the parenchyma become condensed, and its transmitting power thereby improved, the bronchi also becoming better conductors of sound, from the thickening of surrounding parts, the sound of the voice in the thorax is louder, constituting "*bronchophony;*" sometimes a tolerably distinct articulate sound is heard, which is called "*pectoriloquy.*" If the sensory nerves of the ear perceive an unpleasant jarring sensation from the thoracic wall, we have the "strong bronchophony," which therefore in part means that the ear when laid upon the chest feels an increase of the pectoral fremitus. Sometimes the voice as heard within the chest has a nasal, bleating tone, for which phenomenon (*ægophony*) there is no satisfactory explanation. Like the bronchial breathing sound, bronchophony ceases while the tubes are obstructed by secretion, and while their communication with the trachea is interrupted. During the process of resolution of pneumonia, moist *râles* are heard. Sometimes, when the air again begins to enter the minuter bronchi and vesicles, the *râle* is extremely fine, but, as the secretion has less viscidity than before, the sound is not so "dry" as that "crepitation" heard during the stage of engorgement. This sound is called the *crepitatio redux*. The *râles* produced in the greater bronchi, under conditions like those under which bronchial respiration and bronchophony arise, may become bronchial "*consonant*" (*Skoda*) and "*ringing*" *râles* (*Traube*).

The pleuritis which constantly accompanies pneumonia is not susceptible of physical demonstration, excepting when it causes a copious effusion. There are scarcely ever any audible friction-sounds in the first stage of pneumonia, since the pleural surfaces rub together very little, if at all, at that period. They are heard somewhat oftener during resolution, as the air then reënters the vesicles, and the patients breathe with greater freedom, producing friction of the pleural folds.

The physical signs of a great cavity in the lungs, as a result of abscess or gangrene, are identical with those of a tubercular cavity. For a further description of them we refer to Chapter XIII.

DIAGNOSIS.—In children, and in greatly prostrated subjects, particularly in old men, pneumonia is often overlooked. In children this occurs chiefly when the disease sets in with convulsions and a violent fever, attended by very little cough, as little children do not expectorate, nor know how to tell the seat of their pain. Dyspnœa is then attributed to the fever, and, if the child have diarrhœa, the fever is often regarded as a "tooth fever," with inflammatory irritation of the intestinal mucous membrane; or, if the bowels be confined, it may be mistaken for acute hydrocephalus. We ought never to neglect carefully to auscult children with violent fever, brain symptoms, and hurried respiration. The risk of confounding the pneumonia occurring in old and greatly-depressed subjects with typhoid fever is guarded against by the absence of the tumor of the spleen, the eruption, the tenderness in the ileocæcal region, the initiatory chill, above all, by the physical examination of the chest.

The differential diagnosis between pneumonia and pleurisy will be more appropriately considered after we have made ourselves familiar with the symptoms and course of the latter.

Valuable for the diagnosis of pneumonia as we have seen physical examination of the chest to be, it nevertheless is not of itself sufficient to prove more than the existence of infiltration and filling of the air-vesicles. The character of the infiltration is to be ascertained from the history of the case.

PROGNOSIS.—The prognosis, first of all, must depend upon the extent of the disease. Double pneumonia is justly regarded as the most dreaded form. The prognosis, however, depends much more upon the accompanying fever, since, as we have seen, exhaustion from fever terminating in general palsy is the cause of death in the majority of fatal cases. An elevation of temperature above 106° F., an increase in the frequence of the pulse above one hundred and twenty beats, renders the prognosis bad.

Pneumonia is an extremely dangerous disease to aged persons and to drunkards, owing to their intolerance of even moderate degrees of fever; and while but a small proportion of middle-aged patients die of it, the mortality from this disease among old people amounts to between sixty and seventy per cent.

Complications of pneumonia with tuberculosis, disease of the heart, Bright's disease, as well as the occurrence of endocarditis and pericarditis, should cause us to fear an unfavorable result.

Among the individual symptoms, the sputum furnishes a clew to the prognosis. The absence of all sputa must, in the beginning, be regarded as unfavorable, as must also the appearance of very dark, brownish-red (prune-juice) expectoration. This signifies a poor state of nutrition and fragility of the pulmonary capillaries, and, as a rule, denotes a cachectic condition of the individual. Very copious liquid œdematous sputa are ominous of evil. Scanty expectoration during resolution of pneumonia, if the dulness continue to disappear, is of smaller importance; but absence of expectoration, accompanied by gurgling sounds in the chest, signify palsy of the bronchi, œdema of the lung, and approaching dissolution. Delirium at the beginning of the disease is a matter of no gravity, and is due to the derangement of nutrition in the brain, or to the high temperature of the blood which flows through the brain. At a later period it often accompanies exhaustion, so that, when it is persistent and intense, it may be regarded as a sign of an adynamic condition, and hence may furnish grounds for alarm. The same is true of the entire train of symptoms which we are in the habit of calling "nervous" (typhoid). It has already been stated that drowsiness, transient twitchings, or palsy, are dangerous signs.

Finally, the prognosis depends upon the sequelæ of pneumonia. A transition from the stage of hepatization into that of purulent infiltration is of far more unfavorable augury than the termination by liquefaction and absorption. The formation of an abscess, caseous infiltration of the exudation, and gangrene, make the prognosis more and more grave.

TREATMENT.—The indicatio causalis cannot be met in the majority of cases, inasmuch as almost every pneumonia arises from unknown atmospheric or telluric influences. Indeed, it would be highly injudicious to treat a patient with pneumonia by diaphoresis, under the assumption that he had "taken cold."

With regard to the indicatio morbi, we must not forget, in the first place, that the natural course of pneumonia is more decidedly cyclical than that of almost any other disease, and that, left to itself, in a vigorous patient, if uncomplicated, and of moderate intensity, it almost always ends in recovery. This fact has not been known until recently. We have to thank the so-called expectant mode of treatment of the Vienna school and the success of the homœopaths for this important discovery, from which the following rules are to be drawn. Simple pneumonia attacking persons previously in good health requires no more active treatment than does erysipelas, small-pox, measles, or other diseases of cyclical course, provided only that the extent of the disease be moderate, and that there be no complication. Indeed, it has been proved that, unless warranted by special indications, active interference has an unfavorable effect upon the course of pneumonia; and *Dietl* is right in affirming that this disease, when treated by bleeding, more often terminates fatally than where no venesection has been employed. It is quite a different matter to compare the cases in which we bleed, *not because of pneumonia, but in spite of pneumonia*, and for fear of certain complications, with those cases in which, upon principle, blood-letting is never practised. Highly as I prize venesection, however, in certain emergencies which may arise in the disease, I had rather that any one, dear to me, and sick of pneumonia, were in the hands of a homœopath than in the hands of a physician who thinks that he carries the issue of the malady upon the point of his lancet.

The number of bleedings which used to be practised by *Bouillaud* and other disciples of the "*saignée coup sur coup*" school, likewise tends to support the experience of *Louis*, *Dietl*, and others, that bleeding is no specific, and that it does not even cut the process short. In fact, the bleedings had to be repeated and continued until the third, fifth, or seventh day—that is to say, until the terminal day arrived—when the cycle of the pneumonic process was complete.

Whichever one of the current theories upon inflammation we may adopt, none of them even partially upholds the efficacy of venesection in pneumonia. The fact is, unfortunately, forgotten, that the most intense hyperæmia, by itself, cannot occasion croupous inflammation; that the enlargement and dilatation of the capillaries, which we see in valvular disease of the heart, although they may cause splenification and œdema, never produce croup of the air-vesicles. The subject of venesection can be more appropriately discussed while considering the symptomatic indications for treatment, under which head it, strictly speaking, belongs.

I have made extensive employment of cold in the treatment of pneumonia, and, relying upon a large number of very favorable results, can recommend this procedure. In all cases I cover the chest of the patient, and the affected side in particular, with cloths which have been dipped in cold water and well wrung out. The compresses must be repeated every five minutes. Unpleasant as this procedure is in almost all cases, yet even after a few hours the patients assure me that they feel a material relief. The pain, the dyspnœa, and often the frequency of the pulse, are reduced. Sometimes the temperature goes down an entire degree. My patients often retain this surprising condition of improvement throughout the entire duration of the attack, so that their outward symptoms would hardly lead one to imagine the grave internal disorder. The relatives of the patient, too, who do not fail to perceive the improvement, now readily assist in the treatment to which at first they were opposed. In a few cases, and only in a few, the use of cold affords no relief, and the troublesome manipulation for its application increases the distress of the sufferers so much that they refuse to keep it up. In such cases I have not insisted upon the further application of cold.

In the hospital at Prague every pneumonia is treated with cold compresses, and, according to the statements of *Smoler*, it is exceptional for a patient not to feel material relief from this treatment. As, however, I have never succeeded in cutting short a pneumonia by means of cold applications, I should only ascribe a palliative influence to their use, had not the duration of the disease in many instances been decidedly shortened and the convalescence hastened by means of their energetic and methodical employment. In fact, in but few cases have we seen the disease delay its departure until the seventh day. Many have improved on the fifth, and a very large number as early as the third day; nay, I have repeatedly found it impossible to keep patients with recent pneumonia in hospital for a longer period than a week. Cold is rightly regarded as one of the most efficient antiphlogistics in inflammation of external organs. Its action is directly tonic upon the relaxed tissues and dilated capillaries. It is harder to comprehend its mode of action upon inflammation of parts separated from the point of application, by skin, muscle, and bone. However, the contraction of the uterus and intestinal muscles, when cold is applied to the abdomen, proves the possibility of its operation upon the interior, and ice compresses have long and justly been held in repute for meningitis, as have also the cold compresses in peritonitis, by *Kiewisch*. I have no experience of the effect upon pneumonia of the repeated envelopment of the entire body in cold wrappings, as has been practised often by hydropaths; although it may be assumed that it would reduce the temperature of the body, and temporarily moderate the fever, even if it were unattended by any great direct influence upon the local phenomena. At all events, I am able to testify that, in the infectious diseases, active cooling treatment has such an effect upon the elevated temperature of the body in a large number of cases. All other modes of treatment recommended for pneumonia cannot be regarded as addressed directly to the disease, but, like blood-letting, belong to the indicatio symptomatica—being required only when special symptoms arise.

Venesection ought to be resorted to in the following three conditions only: 1st. When the pneumonia has attacked a vigorous and hitherto healthy subject, is of recent occurrence, the temperature being higher than 105° F., and the frequence of the pulse rating at more than one hundred and twenty beats a minute. Here danger threatens from the violence of the fever; and free venesection will reduce the temperature, and lessen the frequence of the pulse. In those who are already debilitated and anæmic, bleeding increases the danger of exhaustion. Should the fever be moderate, blood-letting is not indicated, even in healthy and vigorous individuals. It cannot cut the fever short, and indeed the fever is more apt to persist, although in a somewhat more moderate degree, so that the enfeebled patient is thrown into greater danger than if he had had to pass through a more violent fever, but with unreduced strength.

2d. When collateral œdema in the portions of the lung unaffected by pneumonia is causing danger to life, the pressure of the blood is reduced by bleeding; and, by prevention of further transudation of serum into the vesicles, insufficience of the lung and carbonic-acid poisoning are averted. Whenever the great frequence of respiration in the commencement of a pneumonia cannot be traced to fever, pain, and to the extent of the pneumonic process alone, *as soon as a serous, foamy expectoration appears, together with a respiration of forty or fifty breaths a minute*, and when the rattle in the chest does not cease for a while after the patient has coughed, we ought at once to practise a copious venesection, in order to reduce the mass of blood and to moderate the collateral pressure.

The third indication for bleeding arises upon the appearance of symptoms of pressure upon the brain, not headache and delirium, but a state of stupor or transient paralysis.

Having determined, for one or other of the above reasons, to bleed, the physician must not be misled by the fact that the pulse may be small and feeble instead of full and vigorous. Among the practitioners of the old school, " a small, repressed pulse " was always an indication for blood-letting, and a great number of cases may be advanced to show that the pulse often improves immediately after, or even during the venesection, and indeed the rule obtained that when the physician was in doubt as to whether the debility of the patient was genuine or false, he must take notice whether the pulse became larger or smaller after phlebotomy. The following is the reason why the pulse often grows stronger and fuller during or immediately after a bleeding: *Ceteris paribus*, the size and fulness of the pulse depend mainly upon the ability of the heart to surmount the resistance opposed by the aorta. If the functional vigor of the heart be reduced by the depressing influence which results from an immoderate increase of the animal heat, and which, in some constitutions, is induced by a very moderate elevation of the temperature of the body, the resistance opposed by the aorta remaining unchanged meanwhile, the volume of blood propelled by the heart is diminished and the pulse-wave is small. If, now, we reduce this vascular tension by letting blood, we diminish the resistance of the aorta, and enable the heart, although actually enfeebled, to propel an increased volume of blood, and the pulse rises. True, we may fail in obtaining this effect, when, as sometimes happens, the effect of venesection is greatly to weaken the action of the heart, so that, although we diminish the resistance, we also diminish the propulsive power.

Digitalis has been extensively employed, and with great justice, in the treatment of pneumonia. Like venesection, it is a febrifuge, lowers the temperature, diminishes the frequence of the pulse, without exercising so weakening and depressing an effect upon the system as bleeding. Its exhibition is indicated in pneumonia with a pulse of from one hundred to a hundred and twenty in frequence. With a less frequent pulse it is not required. We usually combine an infusion of digitalis (℈ j— ʒ ss. to ℥ vj) with the neutral salts of nitrate of potassa and soda. If the latter have any influence upon the progress of the disease, it is only upon the fever; they have no antiphlogistic nor aplastic action.

Next come the nauseants (antim. et pot. tart. gr. iv—gr. vj to ℥ vj. S. ℥ ss every two hours) and ipecacuanha; after these, quinine, veratrine, and the inhalation of chloroform. By all of these agents the action of the heart and the temperature can be reduced, and the fever moderated, but they have no immediate local influence upon the nutritive disorder. The use of tartar emetic, formerly so common, has of late fallen somewhat into discredit. According to experiments which I have made in the last few years in the administration of quinine, the indications show that when there is great danger, arising chiefly or entirely from excessive elevation of the temperature of the body, quinine should be given in doses of two grains every two hours, or, what is better, in two or three ten-grain doses within a few hours.

According to the observations of *Biermer*, veratrine (which has already been recognized by *Vogt* as a valuable antipyretic) is one of the most certain remedies for reducing the pulse and lowering the temperature in pneumonia. To obtain its full effect, the twentieth of a grain of veratrine, or the sixth of a grain of resin. veratri viride, should be given in the form of a pill. The results reported by *Biermer* warrant further trial of this article in cases of high fever. Its effects are very variable, and it is apt to occasion vomiting, diarrhœa, great prostration, and collapse.

In the great majority of cases of pneumonia, all the remedies hitherto mentioned may be dispensed with, and the disease will go on rapidly to a favorable termination, under the use of cold compresses and some inert mixture of gum-water, "a tablespoonful to be taken every two hours," ordered to satisfy the patient "that something is being done for him." The more distinctly we keep in mind the particular phases of the malady which call for active treatment, so much the more successful will treatment be.

As the disease advances, the symptoms often demand measures the physiological operation of which is exactly the opposite of all those hitherto described. We have seen that an extensive exudation, a prolongation of the pneumonic fever, or, independently of either of these, a debilitated state of the constitution prior to the attack, may give rise to an acute marasmus, a state of the most complete adynamia, and, indeed, that it is to this exhaustion that most people succumb who die of pneumonia. The feeble contractions of the heart tend to produce new dangers from passive œdema of the lung, and commencing palsy of the bronchial muscles embarrasses the evacuation of the bronchi. Stimulants must now be administered; the heart is to be excited into energetic action; the contractile power of the bronchial muscles must be raised. Fruitless as their extensive employment often is in other diseases, from the transitory character of their action, yet stimulants may produce very gratifying results if given in cases where symptoms of exhaustion arise while the pneumonic process is still incomplete. By giving large doses of camphor, musk, and strong wine, we not unfrequently are able, for a period of about twenty-four or thirty-six hours, to support the action of the heart, to arrest the progress of the œdema, and to facilitate expectoration. The "flores benzoes" (Benzoic acid, gr. v every two or three hours) is particularly recommended for this purpose.

Compensation for waste of substance suffered by the system, which fever and augmented combustion threaten to consume, is of far greater importance than is the use of stimulants. Do not carry the antiphlogistic diet too far, especially in depraved constitutions and in enfeebled persons, but as soon as distinct indications of asthenia begin to appear, in addition to the wine, give concentrated broths, milk, etc.

The bold administration of the preparations of quinine and iron are peculiarly appropriate in these cases. *Rademacher's* tincture of iron is especially applicable (℥ ss to ℥ vj water. S. ℥ ss—two hours). There is no form of pneumonia which, in the sense of *Rademacher*, "eine Eisenaffection des gesammt organismus darstellt," but an impoverishment of the blood often sets in during the disease, the obviation of which is quite as well promoted by the use of ferruginous preparations as is the chronic deterioration of the blood in chlorosis. A physiological explanation of the undoubted usefulness of the preparations of iron in chronic and acute impoverishment of the blood has not as yet been found. We only know that, not only is the iron of the blood increased in amount, but the protein substances, particularly the globulin of the blood, whose quantity always undergoes diminution, increases again under the use of iron. We shall easily convince ourselves that the action of the ferruginous preparations in acute impoverishment of the blood is quite as great as in chronic anæmia, if we use them with sufficient boldness in cases of exhausting pneumonic and pleuritic exudation; and, without assenting to the principles of *Rademacher*, we cannot deny the success which his school has attained by the use of iron in acute febrile diseases. Unfortunately, if diarrhœa exist, they are not well borne.

The employment of stimulants, generous diet, and the preparations of quinine and iron, may be indicated from the very outset of the attack, when an adynamic state develops early, as in the case of old persons, or of cachectic subjects; and it must be regarded as a serious blunder if a physician, who, by his stethoscope, has recognized pneumonia in a supposed "gastric" or nervous influenza, should proceed to treat the malady upon "antiphlogistic" principles. Local blood-letting, by means of leeches or cups, must be resorted to in all cases where the pain is not mitigated by the employment of cold, or when the patient cannot bear, or will not submit to the latter. It almost always mitigates the pain, and as pain is not only a troublesome symptom, but is one of the causes of the disturbance of respiration, its removal may have a beneficial effect upon the progress of the disease. On the other hand, it is better not to employ cutaneous irritants, whether sinapisms or blisters, at all, or at least not until a late period, when resolution is going on too slowly. Finally, if the patient be plagued by cough or restlessness, or by sleepless nights, the indicatio symptomatica may require the use of narcotics, and we must not fear to administer a Dover's powder at night under these circumstances, notwithstanding the persistence of the fever.

## CHAPTER X.

### CATARRHAL PNEUMONIA—BRONCHOPNEUMONIA.

Etiology.—The catarrhal process is a form of disease peculiar to the mucous membranes, and, as no mucous membrane with mucous glands exists in the pulmonary vesicles, the name catarrhal pneumonia is not quite applicable to the disease in question. Nevertheless, as catarrhal pneumonia never arises unless preceded by catarrhal bronchitis, and as its characteristic pathological alterations are entirely analogous to those of bronchial catarrh, we shall retain the generally adopted title. In many cases catarrhal pneumonia arises solely through the extension of the morbid process from the bronchial mucous membrane into the air vesicles. In the great majority of instances, however, this disease develops in pulmonary tissue which has already collapsed, a circumstance which makes it more than probable that collapse of the air-cells essentially favors its occurrence. It is not surprising, moreover, that the capillaries of the alveolar wall, when liberated from the pressure of the air enclosed in the vesicles, should become enlarged and surcharged with blood, nor that after long persistence of this capillary hyperæmia, it should be attended by augmented transudation and copious cell-formation. Now these are the very alterations which the anatomical appearances of catarrhal pneumonia present.

This disease is most commonly observed as a complication of measles, and of whooping-cough; but the reason for this seems to be simply, that capillary bronchitis occurs much more frequently in the course of the latter complaints than in healthy children. Causes of catarrhal pneumonia, other than those from which capillary bronchitis and collapse of the lung originate, are unknown. We may very properly call it a disease of childhood, as it is in children that capillary bronchitis, and its sequel, partial pulmonary collapse—the precursors and initial stages, as it were, of catarrhal pueumonia—are most commonly seen.

Anatomical Appearances.—While croupous pneumonia extends, as a rule, throughout an entire lobe of a lung, or at least throughout a large portion of a lobe, catarrhal pneumonia almost always remains limited to single lobuli, and hence has also obtained the names of lobular, disseminated, insular pneumonia, in contradistinction to the lobar or croupous pneumonia.

If the process have developed in the midst of pulmonary tissue which contains air, we observe in the affected lung distinct scattered firm points corresponding to the inflamed lobuli, which lie chiefly upon the periphery of the lung, and are then distinctly wedge-shaped. Their surfaces lie upon a level with that of the surrounding parts. At first they are of a bluish red; later, if the transudation and cell-growth predominate, they have a lighter and more grayish color. Upon section, the surface presents a smooth homogeneous appearance, and there are none of the granulations characteristic of croupous pneumonia. Upon lateral pressure upon the inflamed spot, there flows over the cut surface an opaque liquid, at first bloody, and afterward pale-gray in color, in which, under the microscope, we may see numerous cells, some of them already in a state of fatty metamorphosis. In a more advanced stage, these inflammatory centres undergo the same changes which we have described as taking place in the spots enclosed in collapsed pulmonary tissue. The gradual transition of atelectasis into catarrhal pneumonia has recently been studied and described with accuracy by *Bartels* and *Ziemssen*. These observers agree in representing that the collapsed portions of lung exhibit alteration of structure even when the collapse is quite recent. In slighter cases, this alteration is limited to the lower sharp edge of the lungs, and to a vertical stripe about two inches wide at their posterior edge upon either side. In severer and more protracted cases, the entire lower lobes of each side are involved, the process sometimes extending as far as the back and inner side of the upper lobes. An attempt to inflate them will succeed; but an unusual amount of force is requisite for the purpose; and the reinflated portion does not resume its former pink color, but becomes of a deep scarlet or vermilion red, a proof that the blood in it has increased considerably in quantity. When the collapse is of long standing, the collapsed parts become more voluminous and resistant, and we find in them separate compact knots of irregular form and size. If we now inflate the lung, these knots remain unchanged while the surrounding parts expand and fill with air. Upon section we constantly find in the centre of these spots a dilated bronchiole filled with tenacious secretion. The cut surface resembles that of the spots of catarrhal inflammation in the uncollapsed lung-substance (see above). In a more advanced stage, the numerous small centres of infiltration often coalesce into voluminous masses of induration, so that a large portion of the posterior part of the lung exhibits a brownish-red, compact, but friable infiltration, out of which only small quantities of purulent but adhesive liquid can be expressed. If the disease be of still longer standing, we find that the color of the dark-brown infiltration has gradually faded from the centre toward the periphery, so that the middle assumes a grayish appearance, its firmness being at the same time materially diminished. Upon microscopic examination, we perceive a further advance in the fatty degeneration of the cellular elements, and a large admixture of granular multinuclear cells (pus-cells). The alterations which we have just described are analogous to those of red hepatization and purulent infiltration with which we have become acquainted as stages of croupous pneumonia; although fibrinous exudation never accompanies the cell-growth in catarrhal pneumonia. Abscesses may form as one of the rarer sequelæ of this disease, while caseous infiltration is a far more common result of this disorder than of croupous pneumonia. Finally, catarrhal pneumonia often results in neoplastic formation of connective tissue with consecutive wasting and shrinking of the parenchyma. At all events, *Bartels*, in a series of cases, in which the disease had run a more chronic course than usual, found, instead of the changes described above, that large portions of the lower lobes had acquired a pale, bloodless, strikingly compact and firm consistence. The cut surface also showed a pale-blue color, and presented a homogeneous, smooth, dry appearance. The parts of the lung thus altered could not be inflated. The bronchi were filled by yellowish caseous plugs. The most striking point was the great increase of the interstitial connective tissue. The condensed portions were traversed by thick grayish-white cords, and bands of connective tissue, which ran in different directions, crossing one another repeatedly, and forming a well-defined network. This termination of catarrhal pneumonia is analogous to the induration which we have described as occurring in croupous pneumonia.

Symptoms and Course.—It is difficult to draw up a comprehensive picture of catarrhal pneumonia, as the disease is never of primary origin, but always supervenes upon a catarrhal bronchitis or a collapse of the lung proceeding from bronchitis, and its only symptoms consist in modifications more or less distinct of the symptoms of the disorder by which it has been preceded. With the exception of the physical signs, which, however, are not always characteristic, the kind and manner of the cough and the character of the fever furnish the most important data for the recognition of the complication which has set in. It is highly suspicious if the sick child fear to cough, or when we find, by its complaints, or, in a very young child, by the distressed expression of its countenance, during coughing, that coughing gives it pain. We have already stated, while speaking of whooping-cough, that the cessation of the protracted coughing-spells and the occurrence, in their stead, of short, harsh, painful "hacks," are very serious symptoms; but, in the catarrh of measles, and in a genuine capillary bronchitis, attentive observers will rarely miss the modification of the cough just mentioned. A fact established by *Ziemssen* is of great diagnostic value, namely, that the temperature of the body always becomes elevated upon the supervention of a catarrhal pneumonia upon a catarrhal bronchitis. While the temperature of the body, according to *Ziemssen*, seldom reaches the height of 102.2° F. in simple capillary bronchitis, upon the development of a catarrhal pneumonia, it often mounts, in a few hours, to 105° F. and sometimes still higher. At the same time the pulse becomes more frequent, the face redder, and the child evinces great terror and restlessness, or, in severe cases, soon falls into a state of apathy and somnolence. Upon examining the chest of a child suffering from measles, whooping-cough, or genuine bronchial catarrh, whose cough has begun to grow painful, or whose fever has suddenly grown worse, or in whom intense fever has arisen where none has previously existed, we must not expect for the first day or two to discover the characteristic physical signs of catarrhal pneumonia. When the pneumonic spots are surrounded by healthy parenchyma, and are of no very great magnitude, neither auscultation nor percussion furnishes any diagnostic data throughout the whole course of the disease. On the other hand, if the complaint have developed from an extensive atelectasis in a few days, an adept in percussion will find a dulness, which is almost always symmetrical, ascending posteriorly upon both sides of the spinal column in a narrow stripe, which is characteristic of it, and which does not extend toward the lateral regions of the thorax until a late period. As the collapsed portion of the lung at first presents but a thin layer, void of air, we must percuss with feeble, short stroke, in order to recognize the dulness. The pectoral fremitus and the respiratory sounds are not as yet altered. At most, the rhonchi and *râles* of the capillary bronchitis, in the vicinity of the collapsed region, are somewhat less loud and less distinctly audible than in other parts of the lung. Should the collapse extend, and should the collapsed part become more voluminous and dense, the dulness becomes more distinct, extends more outwardly, the pectoral fremitus becomes stronger. The breathing is bronchial, any *râles* which may be heard have a ringing character; in brief, the signs of auscultation and percussion are now identical with those of a croupous pneumonia at the stage of hepatization. If not called to see the sick child until this period, it may be difficult and even impossible to decide whether we have to do with a croupous pneumonia or with an extensive catarrhal inflammation of collapsed lung.

(Physical exploration, as we have repeatedly stated, never gives information as to the quality of the condensation of a lung, or of effusion into the pleura.) If, on the other hand, we have had opportunity to observe the progress of the malady from its commencement, the distinction between the two is, as a rule, easy: as the occurrence of double symmetrical condensation and the tardy lateral extension of the narrow condensed stripes indicate collapse of the lung and catarrhal pneumonia; while, on the other hand, a condensation at first confined to one side, and, afterward, spreading over the whole of one of the pulmonary lobes, denotes croupous inflammation of the lung.

The progress of catarrhal pneumonia is sometimes, although not often, a very acute one. The disease may prove fatal in a few days, especially if it attack feeble children. In such an event the countenance, previously red, becomes pale and livid. The lips assume a bluish hue; the eyes grow dull and lustreless; the restlessness gives place to apathy, and to a continually augmenting somnolence. Owing to the serious disturbance of respiration, the pernicious effects of incomplete oxygenation and overcharge of the blood with carbonic acid soon become apparent. It is also rare for a rapid resolution to occur in catarrhal pneumonia, and, even when it does take place, the sudden decline of the fever so characteristic of croupous pneumonia is scarcely ever seen; so that, in doubtful cases, the termination of the attack by a lysis or a crisis may decide the question as to the distinction between catarrhal and croupous pneumonia. It is much more common for catarrhal pneumonia to take on a subacute, and even chronic course. This is especially true of those cases which set in upon a whooping-cough or chronic catarrhal bronchitis. Here, as a rule, not only does the consolidation form slowly and gradually, but it continues stationary with great persistence often for many weeks. The child becomes extremely emaciated, until death finally ensues with the symptoms above given; or, perhaps, after hope has almost ceased, resolution of the infiltration and complete recovery follow.

Tubercular infiltration, abscess, and induration of the lungs following catarrhal pneumonia, present the same symptoms as when they appear as sequelæ of croupous inflammation of the lung.

TREATMENT.—It is easy to understand that if, in the course of capillary bronchitis, the disease extend from the mucous membrane of the bronchi into the air-cells, producing catarrhal pneumonia, the same general directions already given will apply for the treatment of this disease. This is especially the case with regard to local and general blood-letting. According to the recent experience of *Bartels* and *Ziemssen*, the latter never proves of service, and often does considerable harm by reducing the strength of the patient, lowering the energy of the inspirations, and thus tending to encourage the spread of pulmonary collapse; and here I will again briefly call to mind the value of emetics, transitory as it may be, and the frequent lack of success in their use. It has been of great interest to me, that both *Bartels* and *Ziemssen* strongly commend the application of cold compresses to the chest (proposed by me in croupous pneumonia), as by far the most efficient mode of treatment.

## CHAPTER XI.

### CHRONIC INTERSTITIAL PNEUMONIA—INDURATION OF THE LUNG—BRONCHIECTATIC CAVITIES.

Etiology.—The lung, when healthy, has but little connective tissue in its composition. A portion of this combines with numerous elastic fibres to form the pulmonary air-cells; another portion serves to bind together the lobules, while a third belongs to the walls of the blood-vessels and bronchi. There is a large class of cases in which, instead of these mere rudiments of connective tissue, we find large sections of the lung converted into a callous, fibrous mass, the product of a chronic interstitial pneumonia, which must be regarded as one of the most frequent of diseases.

In chronic pneumonia there is no free exudation either into the air-cells, or their interstices, excepting in that form of the affection known as caseous infiltration, of which we shall speak by-and-by, while treating of pulmonary consumption. While in croupous and catarrhal pneumonia the pulmonary tissues themselves suffer little or no nutritive disturbance, in the form of inflammation at present under consideration it is precisely this pulmonary intercellular and interlobular connective tissue which is attacked. The process consists in a hyperplasia of the connective tissue, resulting in an augmentation of the substance of the lung, and in a diminution of its cavities for the reception of air. The newly-formed material, by which the lung is solidified, then undergoes further changes, as do all other neoplastic formations of connective tissue arising from inflammation. At first soft and filled with blood, it afterward contracts, and is transformed into a callous, bloodless substance, occupying a smaller amount of space than was formerly filled by the healthy lung.

Chronic interstitial pneumonia scarcely ever occurs as an independent and primary disease. Even in the interesting cases observed to follow the inhalation of iron or coal-dust, the induration is not a direct result of such inhalation of irritating substances, but only appears secondarily, as a consequence of the bronchitis induced by the irritant:

1. We have seen that interstitial pneumonia is one of the complications of prolonged croupous or catarrhal inflammation of the lungs, and that it results in induration of the latter.

2. Simple collapse of the lung appears sometimes to give rise to an inflammatory proliferation of the interstitial substance, resulting in induration of the lung.

3. The deposit of tubercle, and especially the softening of tuberculous deposits, cancer of the lung, hæmorrhagic infarctions, pulmonary apoplexy, and pulmonary abscess, all produce interstitial pneumonia with "nutritive" exudation (*Virchow*). It is thus that the capsules of connective tissue are found, which separate the products and residue of the processes, above named, from the healthy lung.

4. Interstitial pneumonia not unfrequently forms a complication of chronic bronchitis, when it first involves the parts immediately around the bronchus, but may extend thence, forming extensive solidification of the lung.

The occurrence of bronchiectasis as a result of chronic interstitial pneumonia is easy of explanation. The space created in the thorax by contraction of the lung must be compensated for by atmospheric pressure. The thoracic wall sinks in as far as it is possible for it to yield; but, from the structure of the chest, this collapse is restricted within somewhat narrow limits, so that a vacuum would form within its cavity were it not that the bronchi become dilated by pressure of the atmosphere. This process is usually described as if the contracting tissue of the lung exerted a traction upon the bronchial wall, thus dilating the tubes into spacious canals and extensive cavities. But the extra bronchial traction, which the contracting connective tissue exercises upon the bronchial wall, is not the only cause of bronchiectasis. The discovery of diffuse or sacculated dilatations in the midst of tissue which is simply collapsed, or which still contains air, compels us to ascribe the origin of some cases of bronchiectasis to other sources. Unfortunately, the condition in question is an extremely complicated one; and, in spite of the excellent work of *Biermer* upon the pathogeny and anatomy of bronchial dilatation, its origin is, as yet, by no means satisfactorily explained. We must, therefore, content ourselves by briefly stating that, in some cases, probably the calibre of the bronchus is enlarged by the pressure of stagnant secretion upon its inner surface, especially when the resilience of the bronchial wall is impaired. In other cases bronchiectasis, perhaps, is a result of atmospheric pressure during the act of inspiration, in cases where portions of the lung are incapable of expansion, other portions suffering abnormal compensatory dilatation. In such a case, if the resisting power of the bronchial wall be less than that of the pulmonary substance, or if an obstruction in the smaller bronchi, or other impediment, hinder proper expansion of the vesicles, it would seem that compensatory bronchiectasis may arise in place of pulmonary emphysema.

Finally, it is possible that some bronchiectases may be the result of dilatation of the bronchial wall at points in the upper lobes of the lung, where the tubes yield before the centripetal rush of air driven into them from the alveoli by the act of coughing, and, while giving way before the pressure, cause bronchiectasis instead of emphysema.

ANATOMICAL APPEARANCES.—We rarely have the opportunity of examining interstitial pneumonia before it begins to contract. We then find the pulmonary substance solidified and void of air, in consequence of swelling of the vesicular walls and scanty intervesicular and interlobular connective tissue. At first it is hyperæmic and reddened; afterward of a paler, bluish-gray color. In several cases in which bronchiectatic cavities have been found at the base of the lung in the midst of indurated tissue, I have had an opportunity of observing extensive tracts of pale-red homogeneous substance, composed of young connective tissue, and situated between portions of the lung which contained air.

Products of a later stage of the disease are much more frequently met with. They consist of bands, or irregularly-shaped masses entwined in the pulmonary substance, are of a whitish color, or else are blackened by pigment, and of a dense structure which "cries" under the knife. They surround old masses of tubercle, which have already become caseous, and tuberculous cavities, abscesses of long standing, and the residue of the latter sometimes found in the lung in the shape of calcified concretions. When croupous pneumonia terminates in induration, entire lobes of a lung may become converted into this blackish, callous substance.

In the autopsy of individuals who have worked in coal-mines, or who have inhaled coal-dust in other occupations, the lungs and bronchial glands are often found to be of a deeply-black hue.. From the results of recent investigation, there is no doubt that this coloration depends upon the penetration into the lung of particles of coal. As a rule, the pulmonary tissues sustain this intrusion of coal-dust remarkably well, and there are cases in which this *anthracosis* (that is, blackness resulting from deposit of coal-dust) has been the only lesion found in the lung. In other cases, the black discoloration is combined with an interstitial pneumonia, originating from the bronchial walls, but often extending widely. In other instances, again, cavities are found in the indurated tissue, which are undoubtedly to be regarded as suppurating bronchiectases.

*Zenker*, in a valuable treatise, shows that disease of the lungs may also arise from the inhalation of iron-dust, which, in all essential particulars, is similar to anthracosis, differing merely in the nature of the dust inhaled, and in the color of the lung, which is of a slate-color instead of black. In one of the cases of this disease, reported by *Zenker*, for which he proposes the name of *siderosis*, or of *pneumonokoniosis siderotica* (*!*), the oxide of iron, which had entered the lung, had given rise to extensive induration and to the formation of large cavities.

*Rokitansky* describes saccular dilatation of the bronchi as follows: "We find a bronchial tube widened into a fusiform, or rounded pouch; in the latter case the dilatation often being greater upon one side than another, so that a greater part of the bronchial sac lies out of the axis of the bronchial tube. In rare cases, the size of such a pouch may equal that of a hen's egg. They will often contain a bean, a hazelnut or a walnut. We further find either that any one of the bronchial tubes may become expanded into a pouch of this kind, the tube retaining its normal calibre upon either side of the dilatation, or else quite a large tract of the bronchial ramifications may undergo enlargement. Then, many such sacs of different size are so grouped together that they form, as it were, a vast sinuous cave with many branches, whose individual pouches are bounded and separated from one another by ledges or valvular folds of the bronchial wall."

The inner surface of bronchiectatic cavities is at first smooth, the mucous crypts having flattened out and disappeared through excessive extension. The mucous membrane having thus gradually lost its character, becoming more like a serous membrane, its secretion also at first bears some resemblance to that of a serous sac. We find in bronchiectatic cavities a synovia-like liquid, resembling that found in a greatly over-distended gall-bladder, or in an obstructed processus vermiformis. At a more advanced stage, however, the inner surface often loses its smooth character, and the contents of the cavity undergo change. Owing to the unyielding condition of the surrounding parenchyma, which is not compressed even by the most violent coughing, and particularly if the cavities are situated in the lower lobes of the lung, it becomes extremely difficult to get rid of the secretion. Hence, the latter, exposed to an elevated temperature, and in communication with the atmosphere, is converted into a foul, yellow, stinking ichor, which often acts as a corrosive upon the walls of the cavity, producing sloughs, and depriving the walls of their smoothness. It is not uncommon for severe hæmorrhage to take place when these sloughs separate. In other cases, the putrid contents of the dilated tubes cause inflammation or diffuse putrescence of the lung. In the very rarest instances, the bronchus leading to a cavity becomes obliterated, when its contents may be transformed into a cheesy or calcified paste.

SYMPTOMS AND COURSE.—In its first stage interstitial pneumonia can hardly be recognized with certainty. Should the resolution of a croupous pneumonia be very tardy; if we find, after the lapse of weeks, that the percussion-sound continues dull and the respiration bronchial or indistinct, we may anticipate that the disease will terminate in induration, particularly if the patient have no fever, and gradually recover his health, so that we may exclude the idea of cheesy infiltration. We cannot diagnose the disease with certainty until the thorax commences to sink in at the affected side, and the signs of bronchiectasis appear.

It is quite the same with regard to the interstitial pneumonia which accompanies tuberculosis, and caseous infiltration of the lung. As this is one of the constant complications of the above diseases, we may reasonably infer that the dulness at the apex of the lung observed in consumption is due in part to interstitial pneumonia. The depression of the supra and infra clavicular regions, which sometimes accompanies pulmonary consumption, can only be ascribed to this interstitial pneumonic induration, since neither reduction of the dimensions of the lung, nor depression of the thoracic wall, is produced either by tubercular deposit, caseous infiltration, destruction of the pulmonary substance, or by the establishment of cavities. Although this symptom (which is often erroneously called a pathognomonic sign of consumption) is a very common one among consumptive patients, yet this is only because the process by which the lung is destroyed is almost always accompanied by a chronic pneumonia, which causes its induration and contraction. When chronic pneumonia is associated with chronic bronchitis and emphysema, depression of the thoracic wall is less common. In such cases the only diagnostic signs are the coughing-fits, characteristic of the existence of cavities with rigid walls, and the nature of the sputa.

When the disease is somewhat extensive, signs of dilatation and hypertrophy of the right side of the heart are added to the symptoms above described; and at a later period, where hypertrophy of the heart is no longer capable of counteracting the effects of obstructed circulation, cyanosis appears, with blueness of the lips, puffiness of the face, enlargement of the liver, and finally dropsy, symptoms which, as we have learned, also accompany emphysema. An explanation of this is easy; the obstacle to evacuation of the right heart manifestly proceeding from atrophy of the pulmonary capillaries. We rarely observe cyanosis in the pulmonary induration which accompanies consumption, although in such cases there is a double hinderance to the pulmonary circulation. This is attributable to the circumstance that, simultaneously with the destruction of the pulmonary capillaries, the volume of the blood is reduced by hectic fever.

Easy as it often is to recognize bronchiectatic cavities of the lung with certainty, the diagnosis in other instances is extremely obscure. The signs usually described as pathognomonic of bronchiectatic cavities are only met with in cases which are uncomplicated with tuberculosis or cheesy infiltration, and where the cavities are situated in the lower lobes of the lungs. Bronchiectatic cavities at the apex of the lung, lying side by side with tuberculous cavities, cannot be distinguished from the latter even upon dissection, to say nothing about recognizing a difference between them during life. The manifestation afforded by a bronchiectasis in the lower lobes of the lungs is readily explicable, if we only know the extreme difficulty of discharging the contents of cavities in such dependent positions. The liquid contents of a vomica at the apex of the lung has no difficulty in flowing away through the obliquely descending bronchi, but the discharge from a similar cavity situate in one of the lower lobes, through bronchi whose direction is obliquely upward, is either quite impracticable, or, at least, only practicable while the body is in particular attitudes. (Cases occur in which copious volumes of the thick, yellowish-green fetid contents of a bronchiectatic cavity pour from the mouth of a patient, even before he has coughed, whenever he stoops forward or allows the upper part of the body to sink laterally while lying in bed.)

Owing to the difficulty and incompleteness with which bronchial cavities in the lower lobes of the lung are emptied, and to other unknown causes, the contents of the cavities often undergo putrefaction. This putrid sputum has an extremely penetrating, fetid odor (particularly at the moment of its expectoration), and is less viscid than most catarrhal sputa, often containing caseous plugs, in which clusters of margarine crystals are found. When collected and allowed to stand, it separates into three strata, an upper frothy layer, a middle layer of whitish-gray liquid, and a thick grayish-green sediment; in short, it completely resembles the sputa of diffuse bronchial dilatation, and of putrid bronchitis. Nevertheless, in most cases, it is easy to decide whether we have to do with the latter form of disease, or with a sacculated bronchus. In the former, the coughs follow with short intervals, and all the sputa which the patient ejects are of similar quality. On the other hand, patients with a bronchiectatic sac often announce, of their own accord, that they "have two kinds of cough." Indeed, half a day, or even a whole day, may pass, and the patient will cough but little, expectorating small quantities of catarrhal sputa. This will be followed by a violent paroxysm of coughing, in which, in course of a short time, the patient will eject enormous quantities of putrid secretion. When the fit is over, another long period of exemption begins, the spit-cup remaining empty for six or eight hours, or receiving but a few expectorations of mucus, when another attack will soon fill it to overflowing. The walls of bronchiectatic cavities seem to be tolerably insensible, and the irritation of the putrefying secretion does not appear to give rise to cough. It is only when the sac is completely full, and when its contents reach the neighboring bronchi, which still retain their normal sensitiveness, that the cough begins. We may, therefore, assert that violent coughing-fits, which recur at long intervals, and during which large quantities of putrid sputa are expelled, are pathognomonic of the existence of a bronchiectatic cavity.

In addition to the symptoms hitherto described, there is usually well-marked cyanosis, and, at a later period, dropsy. In bronchiectasis of long standing, I have hardly ever failed to find the clubbed enlargement of the terminal phalanges such as usually forms in cases of persistent cyanosis. These signs of venous engorgement are not, however, directly dependent upon bronchiectasis, being due rather to the concomitant induration of the lungs (see above), and hence they are absent in the very rare instances in which bronchiectasis is not accompanied by extensive induration.

Physical examination always affords very characteristic results when the bronchial sacculation lies close beneath the thoracic wall. When the pulmonary substance about the cavity is consolidated and contracted, the thorax is also depressed at the point corresponding, the percussion sound is exceedingly dull, and the sensation of resistance considerably increased. Upon auscultation, if the patient have not coughed for some time, we hear either an enfeebled respiratory murmur or else indistinct moist *râles*. Upon compelling him to cough, so as to provoke copious expectoration, the enfeebled respiration is often replaced by loud bronchial or even cavernous breathing. On the other hand, there are some cases in which physical examination furnishes no aid to diagnosis, because the cavity is situated more toward the centre of the lung, and is surrounded by normal parenchyma. In spite, however, of the absence of physical signs of a cavity, we may diagnose its existence with positive certainty when a patient, without suffering any precursory dyspnœa, expectorates half a pint or more of purulent secretion in the course of a few minutes. Such enormous quantities of matter could only come from a large cavity, as its presence in the bronchi would render respiration extremely difficult, if not impossible.

DIAGNOSIS.—It is often by no means easy to distinguish a diminution and consolidation of the lung, resulting from interstitial pneumonia, from a similar condition arising from continued compression. The thoracic wall sinks in after either process, and the heart, liver, and spleen are displaced, so that the history of the case is often our sole guide. If it cannot be determined whether the primary disease have been pleurisy or pneumonia, the question often remains unsolved, although bronchiectasis is far oftener a consequence of interstitial pneumonia than of compression of the lung.

In distinguishing bronchiectatic caverns from tuberculous excavations, besides the difference of their situation, the following points are to be taken into consideration: 1. Patients with bronchiectasis are generally free from fever, and hence often long retain a tolerable degree of strength, and suffer but little emaciation. 2. Secondary disease of the larynx and intestine is of rare occurrence in cases of bronchial dilatation; hence, hoarseness and diarrhœa, in a doubtful case, would indicate the tuberculous nature of the disease, although the coexistence of bronchiectasis is by no means excluded. 3. Saccular dilatation of the bronchi is so often accompanied by emphysema that, in forming a differential diagnosis between bronchiectasis and tubercular excavation, the evidence of the existence of emphysema would turn the scale in favor of the former.

PROGNOSIS.—As interstitial pneumonia scarcely ever is an independent affection, the prognosis depends essentially upon the original disease. This is especially the case when the malady accompanies tuberculosis. Extensive wasting of the lung, consequent upon tedious pneumonia, or accompanying chronic bronchial catarrh and emphysema, is often endured for a long time, even after bronchiectatic cavities have formed, the patients only succumbing at a late period, upon the establishment of marasmus and dropsy. At other times, life is suddenly endangered by hæmorrhage from the walls of the caverns, or by pneumonia from diffuse putrescence of the lung.

TREATMENT.—In the stage at which interstitial pneumonia becomes recognizable, it is as impossible to do any thing for its relief as it is to soften and resolve any other form of cicatricial tissue. We are equally powerless to effect the closure and obliteration of bronchiectatic vomicæ. It only remains, therefore, for us to see to the emptying of these cavities, so that the foul secretion may not occasion still greater corrosion of the bronchial walls or parenchyma of the lungs. As a second indication, we must endeavor to limit the secretion, both of the cavity itself and of the bronchi, from which secretion seems to flow into the cavity. Both indications are best met by the inhalation of oil of turpentine, as recommended above. It has already been mentioned that this procedure actually represses the secretion, and we can easily convince ourselves that, after an inhalation of a quarter of an hour, violent coughing follows, and evacuation of the cavities is effected. The inhalations are repeated three or four times daily, and I have seen patients raised by this means from a condition of extreme misery to one of tolerable comfort, which lasted for some time.

## CHAPTER XII.

### GANGRENE OF THE LUNGS.

**Etiology.**—Various forms of mortification have already come under our notice in the foregoing chapters, among others, that of abscess as a sequel of pneumonia, and disintegration of the pulmonary parenchyma as a consequence of hæmorrhagic infaretion. Mortification proper, gangrene of the lung, differs from these forms of necrosis, inasmuch as the dead part putrefies and undergoes chemical decomposition. Putrefactive decomposition of necrotic parts of the economy occurs most commonly in organs which are exposed to contact with the air, such as the skin and the lungs, while in the brain, the liver, and the spleen, as long as they remain within their normal envelopes, putrefaction of dead tissue is not so apt to occur. The transition of necrosis into gangrene is materially promoted if a "ferment" (a bit of putrid material) come in contact with the mortified part. This explains why, though circumscribed gangrene of the lungs may be produced by hæmorrhagic infarction in disease of the heart, through obstruction of the nutrient arteries (the bronchial arteries), it is that such a result is far more common in metastatic infarction, caused by an embolus from some region where putrefaction is going on.

Diffuse pulmonary gangrene arises, in rare instances, during the culminating period of pneumonia, the inflammatory stasis causing the absolute arrest, both of circulation and nutrition, in the inflamed region. Such an occurrence is the more likely when stagnation of the blood in the capillaries causes coagulation of that which is in the bronchial arteries.

Pneumonia caused by entrance into the air-passages of food, or the residua of food, is especially prone to run into gangrene, owing to the putrefaction of these foreign bodies.

Gangrene may also arise with or without previous inflammation, from corrosion of the tissues surrounding a diffuse or saccular bronchiectasis, and their implication in the putrefaction of its contents.

It is difficult to explain the occurrence of diffuse gangrene of the lungs in drunkards, and in persons whose constitution has been much debilitated by misery and deficient nourishment; as is also its frequent appearance in lunatics, even where no foreign body has entered their air-passages, and its occurrence in the course of severe asthenic fever, measles, small-pox, and typhus. It would seem, indeed, as if a part, which already has suffered derangement of its nutrition, were especially liable to die when its tissues are exposed to further inflammatory disturbance (*Virchow*).

ANATOMICAL APPEARANCES.—According to the distinction of *Laennec*, there are two forms of pulmonary gangrene, the *circumscribed* and *the diffuse:*

1. Circumscribed gangrene is the more common form. At isolated points varying in size, from that of a hazelnut to that of a walnut, we find the parenchyma of the lung converted into a bluish-green, moist, frightfully fetid slough, resembling the eschar of the skin produced by caustic potash. It is abruptly limited, and surrounded by œdematous tissue alone. This sphacelous spot, which is at first tolerably firm, and adherent to the adjacent parts, soon decomposes into an ichorous liquid, which merely contains in its interior a somewhat hard greenish-black core, mixed up with rotten and ragged *débris* of the tissue.

The seat of circumscribed pulmonary gangrene is generally the periphery of the lung, and the lower lobes. Not unfrequently a bronchus opens into the gangrenous spot; the ichor of the slough enters the tube, and an intense bronchitis is the result.

In a few cases, the pleura also mortifies; the slough softens, the ichor flows into the cavity of the sac, and thus a dangerous pleuritis is set up; and if the gangrenous centre at the same time communicates with a bronchus, pyopneumothorax may occur. In other instances, diffuse gangrene arises from circumscribed gangrene of the lungs. In very rare cases indeed, interstitial pneumonia arises in the surrounding parts, resulting in incapsulation of the gangrenous point; the sloughs are ejected and cicatrization follows, such as we see in pulmonary abscess.

2. Diffuse gangrene of the lung not unfrequently attacks an entire lobe. We then find the parenchyma decomposed and converted into a putrid, tinder-like, black, stinking substance, saturated with blackish-gray ichor. Unlike the preceding form, the process is not abruptly limited, but is gradually merged in the œdematous or hepatized parenchyma. If the mortification reach the pleura, it too is destroyed. Recovery never takes place, the patient dying of general constitutional disturbance.

Either form of gangrene may lead to introduction of decomposed tissue into the veins, to embolism, and to metastatic abscess in the various organs of the greater circulation.

**Symptoms and Course.**—We have seen that the signs of hæmorrhagic infarction and metastatic deposits in the lungs are very obscure. Even circumscribed gangrene, which develops from hæmorrhagic infarction and metastatic deposits, cannot generally be diagnosed until the gangrenous discharge reaches a bronchus and is ejected. Then, indeed, the foul odor of the breath, the blackish-gray liquid, and also the very ill-smelling sputa, leave no doubt about the nature of the case. Sometimes the fetid smell of the breath precedes the characteristic expectoration by some days. The sputa, like those of the decomposing contents of a bronchiectatic cavity, soon separate into several layers, a frothy superficial one, a liquid middle stratum, and a thicker sediment. The color of the expectoration is of a dirty blackish or brownish color. It contains black, tinder-like masses, and frequently soft cores, containing acicular crystals of fat. In rare cases, it also contains fibres of wavy, elastic tissue. Sometimes, physical exploration affords further information. The sound upon percussion is tympanitic, more rarely dull, and in a few instances cavernous sounds may arise.

Some patients evince the greatest prostration from the beginning; the countenance is "pinched" and livid, the pulse small and extremely frequent, and the patient soon perishes from asthenic (putrid) fever. Others bear this serious disorder wonderfully well. Their general condition is scarcely changed; they walk about, are without fever, and the disease goes on for weeks. In these cases hæmorrhage may arise at a later period, which may exhaust the patient; or, after a time, asthenic fever may develop, to which the patient may succumb, after lingering, now better, now worse, for a long time. Should recovery take place (a very rare event indeed), the odor of the sputum disappears, it gradually becomes yellow, and at last, if the gangrenous spot be incapsulated and atrophied, it may cease altogether.

When diffuse pulmonary gangrene arises from pneumonia, we observe a sudden loss of strength during the progress of the latter disease, with a small irregular pulse, a disturbed countenance, and soon the fetid breath and blackish liquid sputum, with its penetrating odor, are added to the above symptoms. When diffuse gangrene arises independently of pneumonia, it is attended from the outset by signs of extreme adynamia, and by symptoms like those which accompany the entrance of septic matter into the blood, rigors, delirium, stupor, hiccough, etc. The expectoration then often ceases entirely, either because the bronchial mucous membrane itself has become gangrenous and insensible, or else because the patient no longer can respond to any irritation whatever. They now not unfrequently swallow what sputum still reaches the fauces, and thus bring on an obstinate diarrhœa in lieu of the expectoration.

Physical examination in diffuse pulmonary gangrene at first affords well-marked tympanitic sound, and afterward a dull one on percussion. Upon auscultation, we hear indistinct breathing and *râles*, and afterward bronchial or even cavernous sounds.

TREATMENT.—The treatment of gangrene of the lungs is somewhat ineffectual. The inhalations of turpentine, recommended by *Skoda*, deserve consideration, as being recommended by an author distinguished by his skepticism in therapeutics. Whether it be of service in other forms of pulmonary gangrene than those which arise about bronchiectatic cavities, may be doubted. Nourishing diet, wine, infusion of bark, and stimulants may be required by the general condition of the patient. They are of no avail against the gangrene itself, any more than is acetate of lead, creasote, or charcoal.

## *TUBERCULOSIS OF THE LUNG.*

THE term pulmonary tuberculosis continues to be the expression most commonly used to signify consumption of the lungs, a proof that the majority of modern physicians and clinical teachers still adhere to the teachings of *Laennec*, and only recognize one form of pulmonary consumption, the tuberculous form. I have long contested this doctrine, and, upon various occasions, have declared, in direct contradiction to it, that destruction of the pulmonary tissues, the establishment of cavities and consumption of the lung are much more frequently a result of chronic inflammation than of tubercular deposit. And I hope that these views, of whose justness any one may easily satisfy himself who will only study the subject with calmness and without prejudice, will ultimately obtain general acceptation.

The error into which *Laennec* and his disciples have fallen is not that they regard tubercle as a neoplasm, but that they look upon solidifications of the lung, due to entirely different causes, as products of tuberculosis. Even according to modern views tubercle still ranks among the pathological neoplasms, although, however, but one form, the *miliary* form, and one mode of origin, *miliary* tuberculosis, is recognized. It is one of the characteristics of tubercle, that it always appears in the form of small nodules, scarcely as large as a millet-seed, and that the individual nodules never grow into voluminous tumors. The larger so-called tubercular nodules consist always of an aggregation of many small miliary tubercles. All the extensive indurations and enlargements formerly described as *tuberculous infiltration*, or as *infiltrated tubercle*, depend neither upon infiltration of the tissues with tubercular matter, nor upon diffuse development of tubercle, but upon morbid processes of a different nature.

In the lungs it is more especially the residua of chronic inflammation which *Laennec* and his pupils have regarded as tubercular infiltration. The main source of their error was the idea that *caseous metamorphosis*, to which tubercle of long standing almost invariably is subjected, was a specific peculiarity of the disease, and that it might be regarded as a diagnostic mark, by which the tuberculous nature of a growth, wherein the process arose, might be determined. According to such views, the product of chronic pneumonia, which often appears in phthisical lungs independent of tubercle, was ascribable to tuberculosis, since, generally speaking, this inflammatory product at first is moist, transparent, and of a grayish or grayish-red color, and, after a lapse of time, becomes transformed into dry, opaque, yellow, cheesy masses, and, subsequently, into a creamy or curdy, flocculent liquid ("tubercular" pus).

But the point of view, from which caseous metamorphosis was considered a characteristic sign of tuberculosis, is obsolete. It is well established that not only tubercle but many other formations with which it has nothing in common, such as old, cancerous nodules, lymphatic glands enlarged by hyperplastic cell-growth, hæmorrhagic infarctions, incapsulated collections of pus, may all undergo caseous metamorphosis, and the term tuberculization, which has been productive of great confusion, and against which I have long protested, has fallen into disuse.

By this important step in pathological anatomy, for which we are chiefly indebted to *Virchow*, the very foundation of the teachings of *Laennec* is swept away. His fundamental idea that all pulmonary consumption depends upon neoplasm, after having exercised a most baneful influence both upon the prophylaxis and the treatment of the disease, is no longer tenable, and it is really incomprehensible that the majority of physicians of the present day should still adhere to his views.

Although the consolidation and destruction of the pulmonary tissue in consumption is mainly a result of inflammation, yet the frequent coexistence in phthisical lungs of the products of chronic pneumonia and tubercle renders it improbable that the presence of the latter should be purely accidental, and suggests a causative connection between tubercle and the inflammatory lesions. According to the common opinion, this connection is, that tuberculosis is the primary affection, to which the pneumonic process is secondary and dependent. It cannot be denied that this view is right in certain cases; in a great majority of instances, however, the converse is true; the tuberculosis supervening as a secondary process upon a preëxisting pneumonia. It is, indeed, rare for tubercles to form in a lung which does not contain products of chronic inflammation.

As the formation of tubercle never takes place unless preceded by a pneumonia terminating in caseous infiltration of the pulmonary tissue, and, as it occurs with equal frequence, whether the infiltration be a sequel to croupous, catarrhal, or to chronic inflammation, we may assume that there is no direct and immediate relationship, or community of origin, between tuberculosis and the inflammatory disorders which generally precede it, but that their connection is indirect, arising from the caseous metamorphosis of the pneumonic product. The truth of this supposition is materially supported by the fact that, in the rare instances in which tubercles have developed in lungs which were in other respects healthy, caseous deposits have almost always been found in other organs, and no less so by the observation that, in extensive tuberculosis, the oldest and most numerous tubercles are always found in the immediate vicinity of masses of cheesy degeneration. The peculiarly frequent occurrence of tubercle in the lungs is manifestly because there is no other organ in which diseases arise which so often terminate in caseous metamorphosis.

Having thus distinctly stated my belief in a causative relationship between caseous infiltration of the lung and pulmonary tuberculosis, and having called attention to their frequent coexistence, in our next chapter, we may, without impropriety, discuss the subjects of chronic pneumonia with caseous infiltration, and of chronic pulmonary tuberculosis, under a common heading, as the two diseases which play the principal part in pulmonary consumption. In Chapter XIV. we shall speak of acute miliary tuberculosis, which is not accompanied by chronic pneumonia, and which never gives rise to destruction or consumption of the lungs.

## CHAPTER XIII.

### CASEOUS INFILTRATION AND CHRONIC TUBERCULOSIS OF THE LUNGS—PULMONARY CONSUMPTION.

ETIOLOGY.—When pneumonia terminates in resolution, the inflammatory product undergoes fatty metamorphosis; then liquifies, and is absorbed. When the disease is followed by caseous infiltration, the fatty metamorphosis is incomplete. The infiltration dries up; the cells which it contains are atrophied; they lose their rounded form, and shrink, through loss of their water, into irregularly-shaped clots. A large part of this process is evidently mechanical, and it is highly probable that, owing to the immense multitude in which the cells are generated, they become crowded together so densely as to become mutually hurtful, and in consequence waste, shrivel, and degenerate (*Virchow*).

We must emphatically express our dissent from the theory that caseous infiltration of the lung, with its concomitant formation of vomicæ, has its source in a form of primary inflammation of peculiar nature, which is distinguishable from other varieties of pneumonia. The hypothesis of a "tuberculous or caseous inflammation of the lung" is entirely untenable, and only tends to cause fresh confusion. On the contrary, it may be said, with perfect truth, that all forms of pneumonia may end in caseous infiltration under certain conditions, and that there is no form of pneumonia of which caseous infiltration is a sole and constant termination. It is true that the difference is very great in the frequence with which the inflammatory products of the various forms of pneumonia undergo cheesy transformation instead of liquefaction and absorption. In croupous pneumonia such a result is rare; in acute catarrhal pneumonia it is somewhat more frequent, while in the chronic catarrhal form it is almost the rule.

I regard the name chronic catarrhal pneumonia as the only title appropriate to the form of disease usually called infiltrated tuberculosis, and gelatinous or tuberculous infiltration, and which latterly and with equal impropriety has sometimes received the name of tuberculous or of cheesy pneumonia. This lobular infiltration, or (when the disease is extensive, as it often is) this lobar infiltration of the lungs, with its homogeneous section and its color and glitter of frog-spawn, is not dependent simply upon a filling of the air-vesicles with young, spherical cells of indeterminate nature, that is to say, with the anatomical products of catarrhal pneumonia, but arises, with rare exceptions, through extension of a chronic catarrh, with a copious secretion of young cells, into the finer terminal bronchioles, and thence into the pulmonary vesicles. I certainly should attach little weight to the application of the name chronic catarrhal pneumonia to the so-called gelatinous pneumonia, did I not believe that, by calling the disease by its proper name, not only is our comprehension of the etiology and pathology of the malady facilitated, but its prophylaxis and therapeusis are promoted. It is not difficult to understand why chronic catarrhal pneumonia should generally give rise to caseous infiltration, far more frequently, indeed, than the acute form of the disease, or than croupous pneumonia. Owing to the slowness and tedious nature of its progress, the tendency of which is to a perpetual accumulation of young cells in the air-vesicles, perhaps also by an inhalation of cells from the smaller bronchi, thus adding still more to those already generated in the vesicles, the cells are more and more crowded together, thus becoming mutually injurious, and undergoing degeneration.

The knowledge that the majority of cases of consumption are not the result of neoplasm but of inflammation, and that, when tubercles exist in phthisical lungs, the tuberculosis is almost always preceded by a pneumonic process, which, by caseous degeneration of its products, has prepared the soil for the growth of tubercle, has been of material assistance in explaining the etiology of consumption. Numerous well-established facts, which had hitherto defied all interpretation (as long as consumption was always ascribed to neoplasm), are now fully reconcilable to the generally acknowledged laws of pathology.

Predisposition to pulmonary consumption or, to speak more precisely, the predisposition toward pneumonia terminating in cheesy infiltration, is strongest in persons of feeble and delicate constitution. It is by no means meant by this that vigorous persons, possessing normal resisting power against noxious influences, enjoy an immunity from this disease. Indeed, although it is somewhat rare, even croupous pneumonia sometimes terminates in caseous infiltration, with subsequent disintegration of the lung, in individuals who, prior to the attack, were in perfect health, and gave no signs whatever of weakness or delicacy of constitution. In similar manner the most vigorous and blooming children may be attacked by acute catarrhal pneumonia, during the measles or whooping-cough, and may soon perish through caseous metamorphosis of the pneumonic product. The origin of the many deaths which have been observed to take place after an epidemic of measles or of whooping-cough, and which, until recently, has been chiefly ascribed to tuberculosis, is, in most cases, really traceable to the effect of a catarrhal pneumonia contracted during the course of the above-named disorders.

But even a simple, genuine catarrh may extend into the air-vesicles in a person of apparently perfect health and vigor. Healthy men should never feel sure that they will not die of an acute or chronic catarrhal pneumonia, proceeding from a cold, and resulting in caseous infiltration and destruction of the pulmonary substance.

That feeble and ill-nourished persons should be in far greater danger of becoming consumptive than vigorous, well-nourished ones, will not appear extraordinary from this point of view.

Daily experience teaches us that a bad state of nutrition is usually accompanied by a feeble power of endurance of noxious influences. Even without especial knowledge of the fact, it is usually assumed, *a priori*, that feeble, badly-fed persons are "sickly"—that they are especially prone to disease, and that they do not recover as rapidly from its attacks. The frequence with which the various organs of the body are affected by disease differs according to the age of the individual. Persons who, during childhood, have often suffered from croup, pseudo-croup, cerebral irritation, and moist eruptions, are liable during and after the period of puberty to bronchial hæmorrhage and to inflammatory disorders of the lungs.

But delicacy and a liability to pneumonic and other inflammatory disorders are not the only distinctive marks between feeble, ill-nourished subjects and those who are well nourished and strong. All the inflammatory derangements of nutrition occurring in the former class give rise to a very profuse formation of young, indeterminate and perishable cells. It is said of such persons, that their " flesh does not heal," that is, that a trifling wound is apt to be followed by severe irritation, and copious suppuration of the wounded part. This peculiarity is partially attributable to an increased irritability which accompanies constitutional weakness, and partially to the fact that badly-nourished or ill-developed organs, when inflamed, are more prone to the formation of cells of a decrepit and perishable nature, than to the formation of such as are capable of development into new tissue.

The main points of the subject hitherto discussed may, then, be summed up as follows: *The consolidation and destruction of the lungs, which form the anatomical basis for consumption, are usually the products of inflammatory action, and the greater the quantity of cellular elements collected in the vesicles, and the longer the duration of the inflammation, so much the more readily will pneumonia lead to consumption, since these are the conditions most favorable for the production of caseous infiltration. Secondly: pneumonia resulting in caseous infiltration occurs most frequently, but not exclusively, in puny, badly-nourished subjects. This is partially because such persons are especially delicate, and, in part, because all inflammatory nutritive disorders by which they may be affected show great tendency to copious cell-formation, with subsequent caseous degeneration.*

We may now, in few words, define our position with regard to that greatly-vexed question, the relations of scrofula and pulmonary consumption.

It very frequently happens, especially during childhood, that the lymphatic glands participate in this morbid tenderness, which, as a rule, is accompanied by augmentation of irritability and a strong tendency to profuse cell-production. While, in persons exempt from this peculiar tendency, the lymphatic glands neither enlarge, inflame, nor suppurate, excepting in case of intense and malignant inflammation of the parts from which they derive their lymph, very trifling irritants, and mild and innocent inflammation of the region whence the lymphatic vessels originate, suffice to excite the glands, of individuals who are thus affected, into an active production of new cells. Inflammation and suppuration of the glands do not take place in all or even in the majority of cases, the morbid action usually limiting itself to a simple cellular hyperplasia, that is to say, to an enlargement of the glands, from multiplication of their normal cellular elements. But, as the retrogression of all morbid processes in individuals of this class is extremely tedious, the glandular enlargements are exceedingly obstinate in character, and in many instances (and, the greater the mass of cells, so much the more apt is it to happen) a partial or diffuse caseous degeneration of the swollen gland is the result.

Persons whose lymphatic glands participate in the general delicacy of the tissues, and in their tendency to this profuse cell-formation under the stimulus of inflammation, are said to be scrofulous.

We lay especial stress upon the circumstance that, in scrofulous individuals, the tendency to glandular enlargement by cellular hyperplasia is constantly combined with a general tendency to disease, particularly to inflammatory disease. This is so very marked as a rule, that the exciting causes of "scrofulous eruptions," "scrofulous ophthalmia," "scrofulous catarrh," and other so-called scrofulous disorders, are apt to escape observation. It often appears as if such inflammations came on spontaneously ("of themselves," as the laity say). There is no anatomical sign by means of which a "scrofulous" ophthalmia or a scrofulous eruption can be distinguished from similar non-scrofulous disorders, and, with the exception of the implication of the lymphatic glands, it is only from the insignificance of the causes from which the affections proceed, the frequence of their recurrence, and their obstinate persistence, that we can infer their scrofulous nature.

Now, if this feeble power of resisting noxious agents, this susceptibility of scrofulous individuals, have not subsided at the period when the lungs become more especially liable to disease, although the frequence of the moist eruption, the obstinate affections of the cornea and conjunctiva and the like, meantime have diminished, yet pneumonic processes are now apt to occur from causes equally trifling with those which formerly gave rise to the ophthalmia and the eruptions, etc.; and such pneumonic affections evince the same obstinacy which the other so-called scrofulous diseases used to show, a circumstance which greatly favors their termination in caseous degeneration.

Upon glancing over the various causes which experience points out to us as predisponents toward consumption, it will be strikingly apparent that they all agree in one particular, *that they all retard or disturb the normal development and conservation of the organism.*

The tendency to consumption is, in many cases, congenital. When the congenital tendency is due to the fact that the parents were consumptive at the time of begetting the offspring, it may properly be spoken of as inherited. But it is not (as is often asserted) the malady which causes the inheritance, but the weakness and vulnerability of constitution which had already laid the foundation of the consumption in the parents, or which had arisen in them in consequence of that disease. The hereditary constitutional feebleness of the offspring may proceed from other disease of the parent instead of consumption. Parents afflicted by other exhausting maladies, or who are ruined by debauchery, or who are far advanced in years, are quite as liable as consumptive parents to beget children who come into the world with a predisposition to consumption.

Among the influences by which a liability to consumption is acquired, or by which a congenital predisposition to it is aggravated, that of an insufficient or improper diet stands first. Feeding a suckling-babe with bread, pap, etc., instead of the mother's milk, may sow the seeds of the malady. An erroneous regimen is often kept up throughout the entire period of childhood. The child is ill-fed ("verfuttert") as the laity say, and consequently acquires a feebleness and susceptibility to disease identical with a scrofulous predisposition. The comparatively greater prevalence of consumption among the poor than among the more well-to-do classes is in great measure dependent upon the wretched diet of the former, which consists chiefly of vegetables. [Germany.] This also accounts for the increased frequence of consumption, according to the size of towns, or, what amounts to the same thing, the number of its pauper population. Hunger and want, as is well known, are less common in the country than in great cities. The influence of a want of fresh air is quite as baneful as is that of an insufficient or improper supply of nourishment. We have no satisfactory explanation of the mode in which continuous sedentary life, and especially an abode in a close atmosphere charged with effluvia, produces its pernicious effect upon the organism; but the fact has long been established that both scrofula and consumption are far more common in asylums for foundlings and for orphans, in houses of correction, prisons, and among factory operatives who spend the entire day at work in a close room, than among persons who take much exercise in the open air. The objection, that the prevalence of scrofula and consumption in such institutions proceeds from other causes than lack of fresh air, is untenable. The average diet of the populations of many poor villages is much worse, and the number of prejudicial influences far greater, than is the case among the occupants of prisons and houses of correction, and yet they are not equally subject to these diseases.

Not unfrequently persons born with vigorous constitutions, and who have been well nourished, evince a decided tendency to consumption from the effect of some other disease, whereby the prehension or assimilation of their food is prevented, or which is undermining their health in some other way. Many patients with ulcers of the stomach, with strictures of the œsophagus, lunatics who persistently refuse their food, finally die consumptive. In like manner persons afflicted with diabetes mellitus, obstinate chlorosis, or tertiary syphilis, ultimately die of pulmonary phthisis. Among acute disorders typhus is especially apt, when protracted, to leave behind it a predisposition to this disease.

To these predisposing causes, acquired through other affections, may be added those which are provoked by persistent suckling, onanism, venereal excess, by depressing or exciting mental influences, immoderate study, and inconsolable grief. (*Goethe*, in his "Clavigo," instead of making Marie Beaumarchais die of a broken heart, very properly makes her pine away, cough up blood, and sink under consumption.)

I regard the wide-spread doctrine that consumption is solely dependent upon a diathesis, from which it proceeds independently of all so-called "exciting causes," as equally gratuitous and dangerous. The circumstance, that the admission of the origin of this disease from external irritation stood in direct conflict with a theory which no one dared to gainsay, has manifestly prevented an unbiassed interpretation of facts. The deliberate assertions of *Laennec* and his pupils, that "catching cold," and other irritation, had no influence in producing pulmonary consumption, and that it never arose from a neglected catarrh, has had the most pernicious effect, both upon prophylaxis and treatment of the disease; and it is fortunate that the laity, when they suspect a tendency to consumption, are more cautious than would be necessary if what is almost everywhere taught in the lecture-rooms were true.

The exciting causes which may give rise to consumption, where predisposition to it already exists, consist, as I believe, in all influences capable of producing fluxionary hyperæmia of the lungs and bronchial catarrh. We therefore refer to what has been stated already as to the etiology of the latter.

The production of consumption by taking a cold drink while the body is heated must be looked upon as fabulous, or, at all events, as among the ill-explained facts. It is not the cold beverage, but the senseless dancing in the midst of heat and dust by which it is preceded, or else immoderate exertion in running, or some other cause capable of provoking a determination of blood to the lungs, which really is to blame, in the cases when the first symptoms of consumption show themselves shortly after the patient has swallowed a cool draught, while over-heated.

Numerous examples exist in the practice of every experienced physician, in which the cough has commenced on some particular day following a severe cold, soon after which the other symptoms of consumption have made their appearance.

A proof of the extremely important *rôle* played by the presence of foreign substances in the air-passages, as an exciting cause of consumption, is found in the great prevalence of the malady among operatives and other persons who constantly live in a dusty atmosphere, such as stone-cutters, file-grinders, hatters, wool-carders, cigar-makers, etc. Of all foreign bodies which, by irritation of the bronchial walls, and of the pulmonary substance itself, give rise to consumption, the blood which is retained in the air-vesicles and bronchi after a hæmoptysis or pneumorrhagia most frequently has that effect, as we have already explained while treating of bleeding from the bronchi and lungs.

Having discussed the etiology of the pneumonic process which plays the most important *rôle* in the production of pulmonary phthisis, we must now add a few words regarding the etiology of pulmonary tuberculosis.

The development of tubercles in the lung, without the preëxistence of caseous degeneration of the inflammatory products, is less common in chronic pulmonary tuberculosis, which is complicated with chronic pneumonia, and terminates in consumption, than in acute miliary tuberculosis (see Chapter XIV.). The etiology of these exceptional cases is utterly obscure, although it would seem that persons predisposed to inflammation, ending in caseous degeneration, suffer from *primary tuberculosis of the lung*, in the stricter sense of the word, with greater relative frequence.

The caseous masses, upon which the consecutive (secondary) development of tubercles in the lungs depends, are situated, in the great majority of cases, in the lungs themselves, and consist of the products of chronic pneumonia, in a state of caseous degeneration. We have no hesitation in stating that the greatest danger, for the majority of consumptives, is, *that they are apt to become tuberculous*. The conditions which cause tuberculosis to accompany many cases of caseous infiltration with formation of cavities, but not all such cases, and the reason why the complication is sometimes early and sometimes late in its appearance, are at present unknown to us, but it seems that incapsulation of the caseous mass affords a certain degree of protection against tuberculosis.

Next to the caseous products of pneumonia, the exudation of pleurisy and pericarditis in a state of caseous degeneration, and bronchial glands in similar condition, most frequently give rise to tuberculosis.

Under this category the cases may also be included in which the cheesy inflammatory products of the results of tuberculosis of the genito-urinary apparatus, the intestines, mesenteric glands, joints, bones, or superficial lymphatics, are followed by tubercular disease.

In cases of doubt, the discovery of cheesy residua in one or other of these organs may decide the question in favor of tuberculosis. We think it quite possible that, at a time not very far distant, the danger of pulmonary tubercle, which the presence of the cheesy residua of enlarged lymphatic glands produces, will take a place among the indications for the extirpation of peripheral lymphatic tumors, and even for the performance of resections and of amputations.

With regard to the frequence of consumption, it is supposed that from a seventh to a fifth of all deaths are the result of this disease, and that in nearly the half of all cadavers we find traces of the nutritive disorders from which pulmonary consumption proceeds.

During fœtal life, and during early childhood, consumption is rare. Even in the latter years of childhood, bronchial catarrh, with swelling and cheesy metamorphosis of the bronchial glands, or "consumption of the bowels," is far more common than pulmonary phthisis. In the chapter upon scrofula we shall treat at large upon the subject of scrofulous bronchial catarrh, and of the softening of cheesy bronchial glands, and call attention to the liability of such an affection to be confounded with genuine phthisis pulmonum.

Toward the period of puberty, and still more so between the twentieth and thirtieth years, the malady attains its greatest frequence, becoming rarer as life advances, without becoming quite unknown even in extreme old age. Males and females seem to be equally liable.

The former belief in the prevalence of the disease in cold climates, and its comparative rarity in warm ones, is not borne out. There are regions situated far to the north which are well-nigh free from it (Iceland, for instance). *Hirsch*, in his classical hand-book of historical and geographical pathology, declares that the mean temperature due to the geographical and territorial situation of a place has absolutely no influence upon the production or frequence of consumption; that great alternations of temperature and a high degree of moisture favor its development, while in elevated regions its appearance is rare. The rarity of consumption in malarious regions is not constant, and is scarcely due to the influence of the malaria, but is dependent rather upon other causes, such as the paucity of the population and the lack of culture in many regions afflicted by malaria.

Anatomical Appearances.—Upon dissection of subjects who have died of pulmonary consumption, many lesions are found in the lungs, especially pathological cavities—or vomicæ—extensive infiltration and other forms of solidification of the parenchyma, which, when cut into, nearly always exhibit small points which suggest the idea of miliary tubercles.

It is only in rare cases that the diffuse consolidation of the lung-substance presents the granular aspect and other characteristics of a lung hepatized by croupous pneumonia. Far more commonly, there is that homogeneous, dull-looking infiltration with smooth section, which we have described as the product of acute and especially of chronic catarrhal pneumonia. As a rule, the gelatinous infiltration has already undergone the transformation peculiar to cheesy degeneration of inflammatory products. If the latter have but recently commenced, we see, upon the gray or grayish-red dead lustre of the cut surface, a few yellow, lustreless marblings. If the caseous metamorphosis be further advanced, the yellow places are larger, until at last the entire solidified portion of the lung is converted into a yellow, cheesy mass. After the infiltration has become caseous, it may undergo immediate liquefaction, and, together with the tissues, break down into a creamy puruloid matter. Thus cavities filled with the so-called tubercular pus are formed. At last a communication is set up with a neighboring bronchus, through which its contents are discharged by coughing. The walls of these cavities are irregular and interrupted; the pulmonary parenchyma about them is infiltrated with caseous matter, and is in a more or less advanced state of disintegration.

The gelatinous or catarrhal infiltration, which, when attacked by cheesy metamorphosis, and softening of the infiltrated lung-substance, leads to the formation of these cavities, is at first generally confined to single lobules. If the diseased lobule be situated near the surface, the solidified spots bear the peculiar wedge-shape of the peripheral lobules. When seated more deeply within the lung, they form rounded indurations, or, where the process is restricted to the immediate vicinity of separate bronchi, the consolidation runs along the course of the tube. By repetition of the process, and by confluence of many of the lobular centres, a whole lobe or even an entire lung may be solidified and become the seat of vast destruction.

But caseous infiltration of the pulmonary tissues, from whatever form of pneumonia it may proceed, does not in all, nor even in the majority of cases, result in immediate disintegration of the seat of the cheesy infiltration, and in formation of a cavity. Such an event only takes place under peculiar circumstances, and perhaps when the disorder is of extreme severity. It is probably brought about through the crowding together of the accumulated cells in the air-vesicles, whereby they not only encroach one upon another, but exert a pressure upon the surrounding tissues and their vessels, thus depriving the alveolar walls of their nutritive fluid, and causing them to perish and break down. Perhaps the anæmia and necrosis of the pulmonary tissue are favored in severe cases by an extension of the process of proliferation of cells from the surface into the tissues themselves.

If the cell-growth be not of sufficient volume seriously to compress the vesicular walls and their vessels, the caseous masses gradually become still more inspissated, and the shrunken atrophied cells break down into a detritus. Little by little their organic matter disappears, while calcareous salts are deposited until there finally is left a chalky or mortar-like concretion. In other cases, again, the arrested fatty metamorphosis of the cells is reëstablished; they become liquefied, and capable of reabsorption.

While one or other of these processes is progressing in the cellular elements involved in the caseous degeneration, an extensive proliferation of the connective tissue is going on in the lung. The calcified deposits are incapsulated, and the space rendered vacant by the cells, which have suffered fatty degeneration and liquefaction, is filled up by connective tissue. In such cases, the lung-substance does not again become penetrable by the air, but is converted into a dense callous mass; and as the connective tissue, which continues to shrink more and more, occupies less room than the healthy parenchyma which it replaces, the lung becomes reduced in size and the thorax sinks in. But, as depression of the thorax can only take place to a limited extent, the bronchi become dilated into rounded and elongated cavities. This is the most common form of cavity in phthisis where it runs a chronic course. The absorption of the caseous masses, through supplementary fatty degeneration and liquefaction, may be so complete that, upon dissection, we may find nothing except pulmonary tissue in a state of induration from interstitial pneumonia, perfectly void of air, traversed by (bronchiectatic) cavities, and without a trace of caseous deposit. While the apex of the lung usually contains cavities of greater or less capacity, and while a large portion of its upper lobes is solidified —partly by gelatinous or caseous infiltration, and in part through induration and consolidation—upon section of the remainder of the lung which still remains permeable to the air, the small points of induration before alluded to are almost always found projecting above the surface of the cut in the shape of yellow nodules. We must beware of immediately assuming such minute solid spots to be tubercles. Experience teaches that many objects which at the first glance seem to be miliary tubercles, and which were formerly regarded as such, prove, upon closer examination, to be transversely-divided bronchi with caseous contents, or bronchi surrounded by alveoli, with thickened walls and infiltrated with caseous matter. By avoiding such errors in *post-mor-*

*tem* examination, we shall arrive at the conclusion that not a single tubercle exists in very many phthisical lungs, and that consolidation and destruction are solely due to a disorganizing pneumonia.

We believe, however, that *Virchow* goes too far in asserting that the doctrine of miliary tuberculosis of the lung is also almost entirely erroneous, and that nearly all so-called miliary tubercles of the lung are foci of bronchitic, peribronchitic, or pneumonic inflammation. It not unfrequently happens that these translucent grayish nodules which are scattered through the lungs, as well as in most other organs in acute miliary tuberculosis, and of whose tuberculous nature there cannot well be any doubt, are also met with in phthisical lungs. Hence, we must also acknowledge the yellow caseous deposits found in the lungs (notoriously regarded as miliary nodules) to be of tubercular nature, when they coexist with the gray miliary tubercles, and when the latter, together with caseous tubercles, are found in other organs at the same time. There are no means of proving that the caseous nodules are the product of vesicular pneumonia, and not tubercle, as we have no criterion for the distinction between caseous tubercles and caseous miliary nodules of inflammatory origin. I again express my opinion that, exclusive of tuberculosis of the bronchial mucous membrane, the development of secondary tuberculosis in phthisical lungs is of very frequent occurrence.

Hitherto we have been describing the anatomical lesions found in pulmonary consumption, as it occurs in the vast majority of cases, wherein the malady, throughout its whole course, is solely dependent upon chronic pneumonia, or in which tuberculosis does not appear until at an advanced stage of the phthisis, when, although it must be regarded as a most serious complication, it takes but little part in the disorganization of the lungs.

In tubercular consumption, in our acceptation of the term—that is, in the form of phthisis in which destruction of the lung is caused by the breaking down of tubercles, and by secondary pneumonia dependent upon the tuberculosis—the tubercle generally first develops in the mucous membrane of the bronchi, as was first shown by *Virchow*. Even in the trachea and larger bronchial tubes we often find extensive granular patches, consisting of innumerable miliary tubercles, or ulcers with the characteristic marks, according to *Rokitansky*, of primary or secondary tuberculous ulceration. In addition to this, however, in the finer bronchi, besides the evidences of purulent catarrh, we find small whitish or yellow nodules, and, upon examination of a successfully-prepared fine section, we may satisfy ourselves that the development of the tubercle has spread from the bronchus to its lateral and terminal alveoli. According to the line of the section, tubercular groups formed in this manner present the appearance of rounded or wedge-shaped conglomerations of miliary nodules, an appearance rarely or never found in acute miliary tuberculosis where the development of tubercle does not begin in the bronchial mucous membrane. The pneumonic process by which the tuberculosis is attended in tuberculous consumption is much less extensive, as a rule, than that which accompanies the consumption which is solely due to chronic pneumonia, or than the form in which secondary tuberculosis supervenes, at a late period, upon the process of induration and destruction, a circumstance of some importance in the diagnosis of tuberculous consumption. It is also quite an exceptional occurrence for a large part, or, perhaps, an entire lobe of a lung, to become solidified by pneumonic infiltration, nor does caseous infiltration often advance to the stage of induration and contraction. The cheesy infiltration almost always breaks down at an early period, so as to allow cavities to form. It is true that we now and then find the apex of the lung to be the seat of callous induration, or of a deposit of thickened caseous matter, or a bronchiectasis, but it is easy to satisfy one's self that these lesions have no connection with the final disease, and that they are the results of some morbid process of prior date.

As it appears from what has been stated above, the bronchi of phthisical lungs exhibit a great variety of conditions. Gelatinous and caseous infiltration is preceded and accompanied by purulent catarrh of the finer bronchi, with dilatation of their cavity. Disintegration of a deposit of caseous infiltration is ushered in by ulceration of the bronchial wall, and the liquefaction almost always begins in the immediate vicinity of the bronchus. In tubercular consumption, the eruption of miliary nodules appears upon the mucous membrane of the bronchi. The majority of the cavities found in chronic consumption are of bronchiectatic origin, while, on the other side, many of the minuter tubes which traverse the infiltrated and indurated lung-tissue become obliterated. The purulent contents of a closed cavity, resulting from the breaking down of caseous infiltration, are discharged by perforation into a large open bronchus. We not unfrequently see several bronchi, with round or oval mouths, running either squarely or obliquely into such a cavity, but their entrance is always abrupt, and never gradual or imperceptible. Finally, where the bronchial surface has suffered no profound or structural change, it is the seat of a catarrh whose profuse secretion is full of young cells. This bronchial catarrh is the main source of the expectoration of phthisical persons.

Many of the blood-vessels, especially many branches of the pulmonary artery of the infiltrated and hardened tissue, are obliterated. In the walls of cavities the obliterated vessels often form prominent ridges, and sometimes stretch from one wall to the other in the form of ligamentous bridges. It is very seldom that, prior to obliteration of the walls of a vessel, they become so eroded as to cause dangerous hæmorrhage. We shall take this opportunity to call attention to a peculiarity in the circulation of the lungs which frequently arises in phthisis. Many branches of the pulmonary artery becoming destroyed, those of the bronchial dilate, and conduct arterial blood to the lungs. Many newly-formed vessels, springing from the intercostal arteries, also advance through pleuritic exudations into the lung. Thus the phthisical lung receives more arterial blood than the sound lung. Part of it passes into the pulmonary veins, a part into the bronchial veins, and a third portion *passes through the pleuritic adhesions into the intercostal veins.* As the discharge of blood from the cutaneous veins into the overloaded intercostal veins is thereby impeded, they, too, are apt to become overfilled and distended, and a blue net-work of veins appears upon the skin of the thorax. A chronic form of inflammation of the pleura almost always occurs as soon as the affection of the pulmonary substance commences to approach the periphery of the lung. The pleural surfaces become thickened and adherent. The thickening may be so great, especially at the apex of the lung, that it may be covered, as by a cap, with a thick, compact fibrous rind, and at such places it is generally impossible to separate the two pleural surfaces without tearing the lung. In many cases the two pleural surfaces grow together throughout the entire extent of the lung, so that a pleural cavity no longer exists, and so that pneumothorax cannot occur, though the process of destruction advance to the pleura itself. It is only through the rapid disorganization of superficially-seated caseous deposits that perforation sometimes occurs before adhesion is established, or before the adhesions have grown strong enough to prevent air and *débris* of tissue from entering the pleural cavity. In tuberculous consumption, and in secondary tuberculosis, miliary tubercles are often found, both in the pleura itself and in the pseudo-membrane, resulting from the chronic pleuritis. The cavities rarely enlarge in what was formerly supposed to be their most frequent mode of enlargement, that is to say, by caseous disorganization of secondary tubercular deposit in their walls. Generally speaking, no matter in what manner the cavities have formed, their increase in size is the result of a diphtheritic process, an infiltration of their walls, with subsequent decay.

The frequent coexistence of laryngeal disease with pulmonary consumption has been already spoken of in detail. The equally common complication of pulmonary phthisis with ulcer of the bowels, intestinal tubercle, with fatty liver, with amyloid liver, with parenchymatous inflammation, and amyloid degeneration of the kidney, will be again referred to under their appropriate headings.

In recent cases, the right heart, whose outflow is always impeded, is found to be hypertrophied and dilated. In protracted cases, in which the volume of the blood is much reduced, the heart is generally flabby, small, and atrophied. A white coating, like curdled milk, is often found upon the tongue and palate, which microscopically consists of vegetable spores and filaments. The cadaver is usually in a state of extreme emaciation; the skin is thin, remarkably white, and not unfrequently covered with scales of epidermis (pityriasis tabescentium). The feet are often œdematous, and one or other crural vein is frequently stopped up by a thrombus, the corresponding leg being tumefied and dropsical. The entire body is bloodless, excepting the right heart, which, when dissolution takes place gradually, contains tolerably large and soft coagula.

SYMPTOMS AND COURSE.—The course of pulmonary consumption varies in type according as its symptoms are dependent upon pneumonia alone from beginning to end of the disease, or as they become complicated with tuberculosis at a later stage, or are tuberculous from the outset. In most instances these three forms may be distinguished from one another with tolerable precision.

We shall first make a brief analysis of the various symptoms observed in the generality of cases of consumption, with especial reference to the particular morbid process to which each symptom belongs, and shall then endeavor to draw a comprehensive picture of the progress of each of the three main forms of the disease.

Increased frequence of respiration, in greater or less degree, occurs in all forms of consumption, and proceeds from a variety of causes. Moderate acceleration of the rate of breathing is not always accompanied by that distressing sense of shortness of breath requiring continual forced inspiration for its relief, known as dyspnœa. Even patients far gone in the disease often have no dyspnœa at all, excepting when some transient increase of the destructive assimilation going on in the system demands an additional supply of air. While at rest they are fully capable of supplying their blood with oxygen, and of eliminating the carbonic acid formed in the system, without any fatiguing exertion. On the other hand, the increased respiratory frequence may be combined with a severe and persistent dyspnœa, which of course is liable occasionally to still further aggravation, and is one of the most burdensome symptoms of the malady.

The augmented frequence of the respiration and dyspnœa of phthisical patients is due, in part, to a diminution of the breathing surface of the lung, in part to obstruction of the bronchi by the attendant catarrh;

partly, although rarely, to pain during respiration; and partly, and indeed chiefly, to fever. As a rule, dyspnœa is only caused by the joint action of several of these factors. Thus the breathing surface may be excessively reduced in area without the patient's feeling any dyspnœa, and without any acceleration of the breathing while the patient is at rest, provided only that neither severe catarrh, pain, nor fever be present at the same time. Many patients, whose lungs are so much consolidated and disorganized that scarcely half of their capillaries remain to carry on the process of oxygenation, still breathe at the normal rate as long as they sit still or are lying in bed. This is simply because a healthy person, under ordinary circumstances, needs to employ but a very small portion of his respiratory apparatus, in order to obtain his proper supply of air. Nor ought we to overlook the fact that, where the lung is indurated and disorganized, the surviving vesicles are more strongly distended by an inspiration of ordinary depth, and allow more air to escape upon expiration than do the air-cells of a healthy lung. The increased activity of oxygenation which thus goes on in the remaining air-cells manifestly compensates, in a great measure, for the deficiency of those which have perished.

The breathing-surface may be seriously diminished by the presence of miliary tubercles, which, though they may elude physical demonstration, fill up a large number of the disorganized alveoli, and close many of the smaller bronchi. Hence, great rapidity of breathing without dulness on percussion, or bronchial respiration, is one of the most important signs of *tuberculous consumption*, in the narrower sense of the word. If we find that a patient, whose lungs are more or less solidified and destroyed, but who hitherto has suffered but little, if at all, from shortness of breath, begins to exhibit an increase in frequence of respiration and a distressing dyspnœa, there being no increase in the solidification or destruction of the lung or aggravation of the fever to account for it, there is strong reason to fear the addition of a tuberculosis to the phthisis which already exists. Cases arise in which we can infer the existence of such a complication, solely from the disproportion between the small degree of dulness upon percussion and the extreme frequence of the respiration.

It would be superfluous to explain in detail why the respiratory frequence of a phthisical subject is aggravated by pleuritic pain and by exacerbation or extension of the bronchial catarrh, which accompanies the malady, or by its complication with the pleuritic effusion, hydrothorax, pneumothorax, etc. That respiration is accelerated by fever is evident. Fever consists of a morbid increase of calorification whereby the body becomes overheated. The necessity for air is augmented in fever just as it is augmented by every bodily exertion; since, in either process, an extra amount of carbonic acid is formed, and an extra quantity of oxygen is consumed in the system. If we compare the rate of breathing with the elevation of temperature and the frequence of the pulse in phthisis, it will be seen that the want of air is partially relieved by the greater depth of the breaths drawn; for the steep curves which generally are used to mark the character of the very considerable fluctuations of the morning and evening temperature hardly ever correspond to similar abrupt curves representing the rate of respiration. The evening acceleration of the latter seldom exceeds above six or eight breaths a minute, and in many cases is not more than three or four breaths. In some cases no acceleration at all can be detected.

*Pain in the chest and shoulders* is a symptom which is often absent throughout the entire course of the disease. It more commonly accompanies the pneumonic form than the tuberculous form of the malady. In cases where physical examination leaves us in doubt, whether we have to do with tubercle or with small scattered pneumonic deposits, pleuritic pains may be of service both in the diagnosis and prognosis, especially when accompanied by sputa tinged with blood.

Consumption is preceded, in a large number of cases, by a more or less protracted period of cough and expectoration, depending upon the precursory catarrh, which afterward leads to catarrhal pneumonia by extension into the air-vesicles; and, subsequently, to consumption of the lungs by caseous degeneration and disintegration of the inflammatory products. It is highly important to endeavor to ascertain of every patient whether his pallor, fever, and emaciation, have been preceded for some time by cough and profuse expectoration, or whether these symptoms have all appeared simultaneously and before the expectoration became copious. In the first case, formerly ascribed to a postponement of the fever and emaciation until an advanced stage of the tuberculosis (according to *Louis*, this is the case in four-fifths of all cases, while in one-fifth only do the cough and fever begin together), it is more probable, *ceteris paribus*, that the disease is of pneumonic nature, while the latter class of cases are probably of tuberculous origin.

The duration of the precursory catarrh varies. Distinct evidence of propagation of the process into the alveoli and of incipient phthisis is sometimes discernible as early as the second or third week. Most of the instances in which consumption is an immediate consequence of measles and of whooping-cough are of this kind, as well as those in which tuberculosis develops under the disguise of a catarrhal fever or influenza. Conversely, a catarrh may have existed for months and years, growing worse in winter and improving during the summer, and may ultimately attack the air-vesicles. In such cases the physician is often entirely at his ease about his patient, as, in spite of the cough and expectoration, the latter has no fever, maintains his strength, and is in a fair nutritive condition, when the scene suddenly changes and the symptoms of consumption appear.

There is also some variety as to the original seat of this treacherous catarrh. Sometimes it is situated in the smaller bronchi from the very outset; but it by no means rarely commences in the larynx or trachea, whence the process gradually extends into the air-vesicles. The frequence with which such cases occur is shown in the following description by *Andral*—an unqualified adherent of *Laennec's*—from the fourth volume of his "Clinique Médicale:" "The phlegmasia of the air-passages, the symptoms of which precede those of tubercle, does not always commence in the minuter bronchial ramifications, nor even in the greater bronchi. So far from it, indeed, that we have more than once found its point of departure to be in the upper portion of the air-passage, and to consist at first of a simple laryngitis. Persons of this class (who are to be distinguished from those in whom laryngitis only supervenes at a more advanced period of tubercular consumption) do not at this time present any symptoms whatever indicating disease of the lungs, until they are attacked by an angina, which at first seems of but little gravity. The voice, however, remains hoarse; the larynx is the seat of a feeling of uneasiness rather than of pain. Sooner or later, the cough returns in more fatiguing paroxysms. The unpleasant sensation formerly confined to the larynx, now extends successively to the trachea and to the bronchi. Each fit of coughing occasions an unpleasant pricking sensation and a disagreeable feeling of heat, and sometimes a genuine pain beneath the sternum. Thus we may, in a measure, follow the phlegmasia, step by step, in its progress from the organs of deglutition through that of the voice and through the trachea to the bronchi and their ramifications. It is only at this period that the malady assumes a graver aspect. Circulation becomes disturbed, nutrition is impaired, and it becomes evident that tubercles have developed in the pulmonary parenchyma."

In badly-nourished, delicate persons, the extension of the catarrh into the air-vesicles and caseous metamorphosis of the infiltration are more to be dreaded than they are in well-developed and robust ones. Individuals are especially threatened whom we know have often suffered from a catarrh, and whose previous catarrhs have been of very protracted character.

Finally the sputa expelled duing the precursory catarrh sometimes furnish a means of estimating the danger. It is a bad sign if the sputa contain sharply-defined, deep-yellow streaks; for it shows that the catarrh is seated in the finer bronchi, and that its product is full of cells. It is when thus situated and of this character, that its implication of the air-vesicles is most to be dreaded. According to most clinical teachers and physicians who do not share our views, and who ascribe all consumption to tuberculosis, this "expectoration streaked with yellow" (*Louis*) is of course regarded as a symptom of an incipient phthisis or tuberculosis, instead of a sign of a mere preliminary catarrh.

Absence of cough and expectoration during the disease itself is very rare; yet instances are met with now and then, in which infiltration of the lung and caseous degeneration have taken place without previous or concomitant disease of the bronchial mucous membrane. Such patients, at first, often have neither cough nor expectoration. Fever, general malaise, loss of appetite, debility, and emaciation, form a group of symptoms which are often difficult to account for, until physical examination reveals the actual condition. When the pulmonary disease is accompanied by intestinal consumption with violent diarrhœa, the cough and expectoration may decrease or cease entirely, even in advanced stages of consumption of the lungs. This may in some degree be ascribed to the derivative action from the bronchi, caused by the intense intestinal irritation.

A hoarse or inaudible cough is one of the chief signs of tuberculous consumption, or of the complication with tuberculosis of a consumption originating in destructive inflammation. The exceedingly interesting cases in which alteration of the voice or of the tone of the cough of phthisical patients, results from palsy of the vocal chords, from pressure upon the recurrent nerve by indurated pleuritic membranes, are of extreme rarity, in comparison with the instances in which similar symptoms arise from tuberculous ulceration of the mucous membrane. The non-occurrence of hoarseness and inaudibleness of the cough, until an advanced period of the malady, is indicative of consecutive tuberculosis. On the contrary, if the cough have been hoarse from the beginning, especially while the sputa were still viscid and transparent, and before physical examination showed any irregularity, the existence of primary tuberculosis may be suspected. As we have said already, tuberculosis often begins in the trachea or larynx, and only extends into the finer bronchi at a later period.

Although the sputa of consumption are mainly the products of the catarrh which complicates the disease, yet they may exhibit certain peculiarities which serve materially to help the diagnosis.

We thoroughly indorse the assertion of *Canstatt*, that it is a most suspicious sign, and one highly calculated to awaken our apprehension of tuberculosis, when the sputa of a persistent cough, accompanied by fever, long retain the crude character of the expectoration of acute bronchitis. The development of tubercle in the bronchial mucous membrane is generally attended by precisely such obstinate and distressing cough, and by that scanty sputa which contains few organic forms, the "sputum crudum" of the ancients, the "purely mucous sputum" of more modern writers.

Should microscopic examination reveal that the deep-yellow, sharply-defined streaks above referred to contain elastic fibres, recognizable through their arrangement and curve as belonging to the air-cells, we know that the event has happened which the appearance of such sputa would lead us to dread. The profuse formation of cells has extended from the surface of the bronchial mucous membrane into its walls, and the parts surrounding. *The discovery of such elastic fibres is a sure sign of phthisis.*

The intimate admixture of blood with the muco-purulent sputa, whereby the latter acquire a uniform yellowish-red color, is pathognomonic of chronic pneumonia, and we have good grounds for inferring, from the appearance of such sputa in the course of a chronic catarrh, that the air-vesicles have also become involved.

When cavities have formed in the lungs, a peculiar form of expectoration appears, which is generally described as pathognomonic of consumption, and is often and erroneously supposed to be characteristic of tuberculosis. Rounded, numulated grayish masses are found in the spit-cup, separated one from another, by a greater or less quantity of clear bronchial mucus. If the sputa have been collected in a somewhat deep glass, we see irregularly-rounded opaque lumps, having a ragged outline, sink slowly to the bottom. These *sputa globosa fundum petentia* of the ancients are an almost positive indication that cavities exist in the lungs. Under the microscope the lumps are found to consist of young granular cells, showing evidence of fatty metamorphosis, together with a very considerable quantity of irregular angular bodies, and granular detritus. They also often contain elastic fibres from the walls of the air-vesicles. Their opacity and greenish-gray color are due to the unusual amount of definitely-formed solid constituents which they contain, which has been incorporated with them during their long sojourn in the cavity. The rotundity of form is owing to the general tendency of the sputa, after their ejection, to preserve the shape of the space in the lung whence they have been expelled. They tend to sink to the bottom of the vessel containing the bronchial secretion, because but little air becomes mixed with them in the cavity while the bronchial secretion, being agitated by the inspiration and expiration of air, encloses numerous bubbles, and is of lighter weight. The small, rounded, ill-smelling fragments of caseous matter sometimes found in the expectoration, and which are often thought to be actual tubercles by the laity, consist almost always of thickened secretion from the tonsils, although now and then they are small diphtheritic sloughs from the walls of the cavities.

Chemical examination furnishes no test for the distinction between the sputa of simple bronchial catarrh and that of consumption.

Fever is one of the most constant symptoms of consumption both in the tuberculous form and that which proceeds from chronic pneumonia. *Ziemssen* has demonstrated that in children the invasion of the air-vesicles by catarrh is always accompanied by considerable elevation of temperature and acceleration of pulse. This is equally true of the commencement of catarrhal pneumonia in adults. The statement of *Louis*, that, in the majority of cases of tuberculosis (four-fifths), fever only arises at a more or less advanced stage of the malady, is based upon the observations of that author, taken from *Laennec's* point of view, according to which the precursory catarrh itself is due to the presence of tubercle. We have repeatedly called attention to the dangerous consequences of this error, and believe that we may declare that, by precise observation of the temperature and the frequence of the pulse, and by the most careful treatment of all cases, in which fever arises during the course of a protracted catarrh, the development of pulmonary consumption may often be averted.

Not only is the fever an important sign of the extension of the catarrh from the bronchi to the air-cells, but its continuance furnishes the main evidence that the pneumonic process has not subsided. The curves, by means of which we represent upon paper the morning and evening fluctuations of temperature, usually show a wonderful degree of similarity, and we may infer the existence of a consumption from them with the same certainty with which we diagnose abdominal typhus or pneumonia. The difference between the morning and evening temperature is about a degree and a half or two degrees Fahrenheit; very seldom less, and frequently much more. In the morning the temperature is often almost normal, while in the afternoon and evening it may rise to 102° F., or even higher. Such fluctuations of temperature are not peculiar to all kinds of exhausting fever. Upon comparison of the thermal curve of a patient with pulmonary consumption with that of one who is suffering from a tedious peripheral caries, a great difference will be observable, particularly in regard to the regularity of the morning remission and evening exacerbation. After extended study of the hectic fever of phthisis, especially as to the cause which interrupts its regular march, we have as yet come to no conclusion upon the subject. We may mention, however, that the variations in the morning and evening temperature of a true tubercuarrow sense of the word), and of a tuberculosis superven- ic pneumonia, are generally much less. Hence, if the tting fever approaching the intermittent type, the prog- e better than if the fever assume a more continued form. rmer case we have often succeeded in moderating, or even in letely allaying the febrile action, and thereby greatly improving e strength and nutritive condition of the patient; but we cannot claim any such results where there was no morning remission. When the caseous masses become incapsulated, or liquefy, and are absorbed, the fever may cease altogether. Patients are often seen who have large cavities in the apices of their lungs, but no fever whatever. In such cases (the pneumonia having resulted in induration) the physical signs presented, and the globular masses of sputa, which the patient spits up morning after morning, form a striking contrast with his apparent good health, his fresh, vigorous look, his nutritive condition and strength. We have already shown that, in spite of their partial recovery, such persons are still liable to die of consumption, either through recurrence of the pneumonia or through consecutive tuberculosis; and we would advise that the condition of the patient as to weight and temperature be still kept under observation, that we may be apprised of it, in case either event occur. We see, then, that, in the diagnosis, prognosis, and treatment of consumption, the use of the thermometer is as great as, if not greater than, in any other disease.

The subjects of *emaciation and deterioration of the blood*, the symptoms to which consumption owes its name, properly succeed that of fever, since there can be no doubt that it is to fever that they are mainly due. A most striking proof of the soundness of the theory, that the elevation of temperature in fever is dependent upon an increase in the calorification, consists in the rapid loss of weight which the body sustains even in a fever of short duration. For years, at my clinic, the fact has been established, by dint of innumerable measurements and weighings of consumptive patients, that their loss and gain in weight stood in direct proportion to the increase or diminution of their fever. There is a very pretty theory, that a continued fever of moderate intensity consumes less (especially if the patient keep his bed) than a hectic fever like that of phthisis, in which the temperature fluctuates daily between a condition almost normal and one of a considerable degree of intensity. There is no doubt that both calorification and consumption of the constituents of the body go on with great rapidity during the rapid rise in the temperature, as has been proved by *Immermann*, but we still hesitate to accept the absolute truth of the above hypothesis. Knowledge of the fact, that it is the fever which consumes both the strength and substance of phthisical patients, is a matter of the utmost importance in the treatment of the disease.

*Physical Signs.*—Inspection of the chest reveals the existence of a "phthisical habit" in many persons who suffer from phthisis, or who are threatened by it. This term is used to signify that peculiar build of the body indicative of a want of proper nutrition and development, and which is found in persons who have been subjected to debilitating influence capable of stunting the healthy growth of the system before their bodies have become fully developed. The bones of such persons are slender, their skin is thin, their cheeks have a delicate redness, the sclerotica is bluish, the subcutaneous connective tissue contains but little fat. The muscles are ill-developed; those of the neck allow the thorax to sink, causing the neck to seem too long. The intercostal muscles permit the ribs to spread widely apart, making the intercostal spaces broader; the angle at which the ribs are attached to the sternum is acuter; the entire chest is flatter, narrower, and longer than in robust, muscular persons. The shoulders also are apt to sink forward, and the inner edges of the scapulæ are tipped up like wings.

The diagnostic and prognostic significance of the phthisical habitus has been a good deal underrated of late, and, no doubt, many persons possessing such a conformation do live exempt from phthisis and attain a good old age. But such a circumstance does not in the least conflict with the belief that the phthisical habit is a valuable index of feebleness and delicacy of constitution, hence of a tendency to consumption. There is greater danger that a catarrh at the apex of the lung will invade the air-vesicles in a patient of this kind, than in a muscular and robust man.

Depression of the supra and infra-clavicular fossæ upon one or both sides, which, hitherto, has always played a great *rôle* in the symptomatology of phthisis, is indicative neither of tuberculosis, nor of caseous infiltration, nor of disorganization of the tissues; but is always and solely due to decrease in size of the apex of the lung, by induration and shrinking. As this is the only process capable of causing depression of the thoracic wall, the symptom is rather a favorable sign, indicating a comparative cure of the nutritive derangements which are the chief causes of consumption. We are not warranted, therefore, in forming a diagnosis of consumption, unless signs of an advancing destruction of the lung be also present, besides the symptom in question.

Feebleness of the respiratory movement, when it corresponds to a depressed point in the chest, is of similar import. In such a case the contracted lung is impermeable to air, and cannot yield to the traction of the inspiratory muscles. If the spot, which remains stationary upon inspiration, retains its normal convexity, the percussion-sound over it, however, being dull and flat, we may infer the existence of an extensive solidification of the lung, which is most probably a pneumonic infiltration. A feeble respiratory movement at a point where the percussion-sound, instead of being dull, is normal, or somewhat hollow and tympanitic, is a suspicious sign of tubercle, but not a conclusive one, as small scattered spots of lobular pneumonia may also weaken the movements of respiration without causing any dulness upon percussion.

There is often an unusually wide extension of the shock of the cardiac impulse, and an outward dislocation of the apex of the heart, when the upper lobe of the left lung is indurated and contracted, thus laying bare the pericardium and drawing the heart to the left. This symptom, like depression of the thoracic wall, denotes a partial recovery from the pneumonic process, and a patient is not to be pronounced consumptive unless it be accompanied by fever, loss of flesh, or other sign of inflammatory or tubercular destruction of the lung.

*Palpation*, besides being serviceable in estimating the movements of respiration, and the degree of dislocation of the apex of the heart, often exhibits abnormity of the pectoral fremitus in phthisis. Over large cavities, containing air, and communicating with an open bronchus, the fremitus generally is intensified. It is also rendered stronger by lobular infiltration and by extensive tuberculosis, which has occasioned a relaxation of the pulmonary tissue. According to *Seitz*, however, for whose opinion I have great respect, the vocal resonance is of little diagnostic value in consumption.

*Percussion* furnishes several diagnostic points of the utmost importance.

Since *Seitz* first caused me to observe that it was easy to mark out the upper boundary of the lungs, and that this was easier to do in front than behind, and when the mouth is open than when shut (since the tympanitic sound of the trachea is then more definitely distinguishable from the non-tympanitic sound of the apex), I never neglect this mode of examination of patients with chronic pulmonary affections. I can assert that the height of the pulmonary apex, which, under normal conditions, is equal upon each side, and which extends from three to five centimetres beyond the collar bone, is often found to be much lower, especially upon one side, when the lungs are in a state of chronic disease. A depression of the upper boundary, therefore, like the depression of the supra and infra-clavicular regions indicates induration and contraction of the apex of the lung.

A dulness upon percussion, in the supra and infra-clavicular region, extending over the clavicle itself, and posteriorly over the supra-scapular and supra-spinatus regions, is recognized even by many of the laity as pathognomonic of phthisis. Most patients, when they consult a new physician, can state precisely what the size and extent of the dulness was at the last exploration. Dulness in these regions signifies that a large tract of the parenchyma is infiltrated or consolidated by growth of connective tissue. Tuberculosis never gives rise to a consolidation of sufficient magnitude to render the percussive sound dull. Hence, as a general rule, it is a favorable sign when the area of dulness accords with the other symptoms, and when it extends its limits in proportion as the malady advances. If it be otherwise, there is reason to fear the existence of tuberculosis.

The presence of lobular infiltration and of miliary tubercles, by which the lungs' capacity for air is reduced, may give rise to a percussion-sound which is not dull, but hollow and tympanitic. Much more commonly, however, the percussion is not affected by such a condition of the lungs. A distinctly tympanitic sound is most frequently heard over a cavity containing air. If the pitch of the ring be altered by opening and shutting the mouth, it is a sure sign of a cavity.

From the *metallic*, *tinkling* sound upon percussion, which is of very rare occurrence in consumption, it may be inferred that beneath the point struck upon there is a large empty cavity, with smooth, regular, and baggy walls, but we must first make sure that there is no pneumothorax.

The *cracked-pot sound* is produced upon percussion, over the seat of a superficial cavity with thin walls, whereby the air is expelled into a neighboring cavity, or into a bronchus with a hiss, which is characteristic of the "*bruit de pot fêlé.*"

*Auscultation*, at the commencement of the disorder, and indeed often in its more advanced stages, shows no irregularity beyond the signs of a catarrh at the apex of the lung. There is a feebleness of respiration, at other times it is extremely harsh, or the breath may be drawn in a series of jerks (*saccadé*). But, above all, there is the greatest variety of moist *râles* and peculiar squeaking rhonchi. Sometimes, after the patient has coughed, the moist *râles* and the crackling, squealing sounds cease. More frequently they are only heard after the first breaths which follow a cough (*Seitz*). It is, therefore, always advisable, in ausculting a patient, to make him cough from time to time. It is easy to understand why peribronchial and pneumonic deposits, which have not caused much solidification, and why tubercles and tubercular masses, and cavities enveloped in parenchyma, still pervious to air, do not produce other symptoms than those of catarrh; but I must most decidedly express my disapprobation of that prevalent belief, according to which the signs of catarrh of the summit of the lung are pathognomonic of consumption, as being both false and prejudicial to the patient. I certainly regard catarrh of the apex of the lung as a serious symptom, and the longer it lasts, so much the more have we to fear that it may lead, or that it has already led, to those derangements of nutrition from which consumption so often proceeds; but, we are not at liberty to conclude that the catarrh has involved the substance of the lungs themselves, until fever, emaciation, pallor of the skin, the presence of elastic fibres in the expectoration, and other evidences of phthisis arise, besides the catarrhal signs.

Bronchial respiration, bronchophony, and sonorous *râles* are heard in cases of consumption, when extensive induration enclosing large open bronchi, or cavities, has formed near the surface of the lung. Indurations of such magnitude never proceed from tubercle or tubercular conglomeration alone. Whether they are the result of infiltration or of induration, whether they contain bronchi or cavities with air in them, must be determined from the other symptoms. When the cavities or bronchi which traverse the solid part of the lung are filled with secretion, no respiration is audible.

Sounds are heard sometimes which place the existence of cavities beyond all doubt, and which therefore are called *cavernous* sounds. The cavernous sounds include—1st. Coarse moist *râles*, audible over places where there are no large bronchi, where large bubbles can form, as at the apex of the lung. 2d. The sudden transition (called metamorphosing by *Seitz*) from a sharp hissing or sucking sound to bronchial respiration, or into indistinct murmurs and sonorous *râles*. This very common and very characteristic sign is probably produced by the entrance of air into a cavity through an opening which at the commencement of the respiratory act is narrow, but which is enlarged as the chest becomes inflated. 3d. Amphoric breathing, the *râle* with metallic resonance, a sound like the bursting of single bubbles with a metallic ring, the *metallic tinkling*. These noises may be produced artificially by blowing over the open mouth of a bottle, or by agitating a liquid in a bottle held before the ear, or by letting fall a drop into the bottle, the ear being placed against it. It is only when there is a similar condition of the lung, when it contains a capacious cavity with symmetrical concave walls, capable of producing uniform reflection of the sound-waves, that amphoric breathing and metallic sounds are audible.

It but rarely happens that diagnostic information of any value in consumption is obtained by use of the *spirometer*, and by measurement of the capacity of the lungs, that is, of the volume of air expelled from the chest after drawing as deep a breath as possible. There are cases of obstinate cough, where percussion and auscultation, giving negative results, excite the suspicion of the existence of lobular infiltration, or of tubercle in the lung. In Germany, the vital capacity of adult healthy men is about 3,300 cubic centimetres, but it varies according to sex, age, weight, and size, so that, when the stature is between five and six feet, every additional inch increases the vital capacity by about 130 cubic centimetres. But even after making due allowance for all these conditions, there still remains considerable variation, depending upon whether the patient be skilful and practised, or awkward and inexperienced. Hence, although a normal or remarkably great capacity of the lungs indicates that they are healthy, no conclusions can be drawn from a slight reduction of their capacity below the normal standard, and it is only when the decrease amounts to several hundred centimetres, and when it cannot be ascribed to want of skill, or to lack of power, and after excluding all other sources of impediment to respiration, that spirometry can contribute toward the diagnosis of an incipient phthisis.

We shall now endeavor to describe the main features which characterize the separate varieties of pulmonary consumption, beginning with that form in which the symptoms and termination are solely due to inflammatory action.

At the outset it not unfrequently assumes the aspect of an acute disorder, with symptoms of greater or less violence. This is the case when a croupous pneumonia, instead of ending by resolution, passes into caseous infiltration, followed by consumption. It also occurs when the blood effused into and coagulated within the bronchi and air-cells during a hæmoptysis causes intense and extensive pneumonia, as well as in cases of invasion of the pulmonary vesicles by acute catarrh of the bronchi.

In a croupous pneumonia, when the fever persists beyond the end of the first or beginning of the second week of the disease, when it becomes considerably aggravated toward evening, and remits toward morning, with profuse perspiration; when the dulness in the thorax continues, and when moist *râles* still remain audible over the affected region, and when the expectoration is profuse and muco-purulent, it is to be feared that the malady has terminated in caseous infiltration and consumption, which is a somewhat rare occurrence. The discovery of elastic fibres in the sputa, and of cavernous sounds, dispels all doubt that the tissues are in a state of cheesy infiltration and decay. The majority of patients die in a few weeks, consumed by the intensity of the fever. Far more rarely, the malady subsides after exciting the very worst apprehensions; the sputa become scanty, and the patient slowly begins to improve. The dulness, however, remains. The thorax sinks in over the affected region, and, after a while, well-marked evidence arises of induration and contraction of the diseased portion of the lung, as well as of bronchiectatic cavities. The pneumonia which follows immediately upon a hæmoptysis or a pneumorrhagia, and which, in my opinion, is caused by effusion and coagulation of the blood within the bronchi and air-vesicles, is of a very similar character. The greater the area of dulness which develops after an attack of hæmoptysis, and the longer it lasts, the more pronounced the pleuritic symptoms, the more intense and persistent the fever, so much the more reason is there to fear that the retained blood and the inflamed parenchyma have undergone cheesy metamorphosis, involving serious disorganization of the lung. As we have already explained, however, subsequent liquefaction and absorption of the caseous mass are still possible, as are also its incapsulation and induration of the affected lung through profuse proliferation of the connective tissue, followed by contraction.

The invasion of a considerable number of air-cells by an acute catarrh is sometimes attended by such serious symptoms, especially violent fever and a rapid decline of the strength and nutritive condition, that the diagnosis is sometimes difficult. It is excusable in such cases if for a while, and until reliable data can be obtained, the physician ascribe the catarrh and intense fever to infection, or to acute tuberculosis of the lung. The case soon clears up, however. The sputa begin to assume the characteristic admixture of blood peculiar to pneumonia, pleuritic pains, of varying severity and extent, are felt, the percussion-sound becomes hollow and tympanitic in the upper part of the chest, and, if the points of solidification, originally lobular, coalesce into one voluminous mass, the percussion-sound is dull. At the same time the *râles*, which at first were indefinite, become ringing, and the respiratory murmur becomes bronchial. It is possible that an acute catarrhal infiltration may undergo complete resolution; far more generally, however, the infiltrated tissue suffers caseous metamorphosis, and soon disintegrates. Most cases of galloping consumption, where wide-spread destruction takes place in a lung within a few weeks, the patient quickly wasting away and sinking under violent fever, arise from the extension into the vesicles of an acute catarrh, involving a considerable portion of the lung, and which may be called an acute phthisis, resulting from acute or subacute catarrhal pneumonia. When an entire lobe of a lung is involved in a process of this kind, subsequent absorption or incapsulation of the caseous deposit, with induration and wasting of the affected part, rarely occurs. Such a termination is much more frequent where the disease is less extended. We may often trace back a depression of the supra and infra-clavicular region, with sinking of the summit of the lung, to an attack of acute catarrhal pneumonia, which has become chronic, and resulted in induration and contraction. We not unfrequently have the opportunity of observing patients who have survived such attacks again and again, at varying intervals, the area of dulness and depression of the thoracic wall extending itself on each occasion, and who are finally carried off by a tuberculosis, or a less fortunate repetition of the pneumonia.

In contrast to the form of disease hitherto described, the implication of the air-cells in the bronchial catarrh may be unattended by any violent symptoms, and may even be entirely latent. Upon dissection, we often find the apex of the lung to be the seat of cicatricial contractions, of incapsulated caseous deposits, and callous indurations, resulting from a pneumonia which has totally escaped observation. And we find many persons whose supra and infra-clavicular regions are sunken in, and the summit of whose lungs is in a state of abnormal depression, without any clew as to the date of the pneumonia by which the apex of the lung has become solidified and wasted. Still, whenever the inflammatory process is at all extensive, even chronic catarrhal pneumonia is almost always accompanied by fever. True, for a while, this insidious fever is generally unobserved, or else misunderstood by the patient, and sometimes, too, by the physician, as the more obvious subjective febrile symptoms, the shivering, sense of heat, thirst, and the like, are slight, and are thrown into the background by the wasting, and the pernicious influence of the fever upon the appetite, the digestion, the hæmatosis, and general nutrition. When a patient with chronic bronchial catarrh, which has no ill effect upon his general health and activity, begins to lose appetite, to grow pale and thin, and to perceive a marked decline in his strength, there is reason to suspect that the pulmonary vesicles have become involved in the catarrh, and it is our imperative duty to ascertain the existence of fever, and of solidification of the lung, by careful measurement of the temperature, and by repeated physical examination of the chest. The chronic form of catarrhal pneumonia shows a decided tendency, under favoring circumstances, to end in induration and shrinking, as well as to relapse under pernicious irritation. This is the reason why so many persons, in spite of the callosities and bronchiectatic cavities in the summit of their lungs, feel tolerably well during the summer months, and gain in strength and weight, while in winter, especially if obliged to work, and to expose themselves to cold, they grow feverish, thin, and pale, and suffer further induration of their lungs. Such alternations often go on for a number of years. Patients of this class furnish a large contingent to the hospitals, where (unless they present some physical signs of especial rarity) they are apt to be unwelcome guests, "chronic pulmonary tuberculosis," as it is called, being generally regarded as a somewhat uninteresting disease. The striking manner in which this form, which is by far the most common form of phthisis, yields to treatment, and especially to dietetic treatment, in the wider sense of the word, is perfectly comprehensible when looked at from our point of view, and furnishes an argument in favor of the theory.

The development of tuberculosis in lungs which are already consumptive, as a result of inflammatory action, sometimes takes place in a manner so latent as to make it extremely difficult, if not quite impossible, to recognize the fact with certainty. On the other hand, there are many instances, especially where the lungs are the seat of very numerous tubercles, and when the tuberculosis involves other organs, in which the diagnosis presents no difficulty. Where we find a consumptive patient to be growing very short of breath, there being no perceptible increase in the dulness upon percussion to account for it; if, in spite of the most careful treatment the fever continue, and if it change from the remitting to the continued form; should diarrhœa set in in a patient who hitherto has been somewhat inclined to constipation; if hoarseness and aphonia be combined with the other symptoms of consumption, or if signs appear of disease of the meninges of the brain, we may confidently infer that tuberculosis has developed in the already consumptive lung. In young subjects, who are peculiarly liable to tuberculosis of the cerebral membranes, brain-symptoms may aid in forming a diagnosis, while in persons of more advanced years the appearance of intestinal or laryngeal symptoms may do the same.

The development and progress of a *tuberculous consumption* differ essentially in type from any thing hitherto described, and its symptoms are so characteristic that the diagnosis of this form of consumption (which is not common) is, as a rule, easy. In the first place, it has no precursory catarrh. The fever and wasting are not deferred until the sputa become profuse and purulent, the tubercular eruption being accompanied by a marked elevation of the temperature and rapid emaciation of the body from excessive calorification. If we are informed that a patient did not begin to cough and expectorate until several weeks after he had begun to decline in strength, and to grow pale and thin, there is always reason to fear that he has tuberculous consumption. Our suspicion will receive confirmation if the patient be unwontedly short of breath, and if, at first, physical examination of the chest give negative results. At a later period the percussion-sound may grow dull from consecutive pneumonia, the respiratory murmur becoming bronchial, and the *râles* ringing, but the solidification is rarely as extensive as in the forms of consumption previously described.

The sound of the voice and of the cough soon grows hoarse, and if there be much tuberculous disease of the larynx, and if it spread rapidly, the well-known distressing symptoms of laryngeal consumption make their appearance. Nor is it long before the signs of intestinal tuberculosis and intestinal consumption set in. Exhaustion is accelerated by profuse diarrhœa. The abdomen becomes sensitive to pressure. The malady seldom lasts over a few months, and most patients succumb even sooner.

It would lead us too far were we to attempt to make a detailed description of the numerous modifications to which the different forms of consumption are subjected by the manifold changes of acute and chronic disease, and the various intercurrent accidents and complications. I am sure, however, that most cases of consumption which we observe ourselves, or which are properly reported to us, may be assigned without difficulty to one or other of the above-given categories.

It follows, from what has been said, that death is the most frequent result of all forms of pulmonary consumption, and that it is the sole termination of tuberculosis, but that, in the forms of the malady which are dependent upon pneumonia, an improvement and approximative recovery are not as rare an occurrence as is usually supposed. It has been satisfactorily established, moreover, that even persons in whom all evidences of consumption have disappeared, and who are completely well of the malady, are still in greater danger than other persons of dying of a fresh attack of the pneumonic process or of tuberculosis.

The fatal termination usually takes place through gradual consumption, "wasting away," "decline." The emaciation of the patient finally becomes extreme. The skin seems too loose for the body, owing to disappearance of the fat and atrophy of the muscles. The zygomatic bones project from the sunken cheeks, the nose seems longer and more pointed, the orbits from which all the fat has disappeared seem too large for the eyes, the nails become incurved, the pad of fat upon the last phalanges being gone. Not unfrequently the temper of the patient, which at first was sullen and perverse, now grows cheerful and kindly. Many have perfect confidence in their recovery up to the moment of death, and expire in the midst of plans looking far into the future. Toward the last, however, the suffering is often severe. If the larynx be also "consumptive," there is an incessant cough which robs the patient of his rest at night; aphthæ form in the mouth and pharynx, rendering chewing and deglutition difficult; the decubitus causes severe pain; one or both of the feet become the seat of an extremely painful œdema, owing to thrombosis of the femoral vein. In such cases the final stage seems extremely wearisome to the physician and attendants, and even to the patient himself, who often longs for his release.

Very much more rarely consumption results in death from hæmorrhage. This is most usually the consequence of erosion of an unobliterated vessel in the wall of a cavity, or else of an aneurismal expansion of a blood-vessel, which, being so situated in the wall of a cavity as to be deprived of support of the indurated pulmonary substance, yields to the pressure of the blood and finally bursts. In these cases of pneumorrhagia the patient either rapidly bleeds to death or else suffocates, the trachea and bronchi becoming filled with blood, thus cutting off entrance of the air into the lungs. The occurrence of pneumothorax, which we shall describe in detail hereafter, is a more common cause of death than hæmorrhage, as are also secondary degeneration of the kidneys, intestinal phthisis, tubercle of the bowels, pneumonia, pleurisy, and other acute diseases.

TREATMENT.—The treatment of consumption has made great advance since recognition of the fact that the disease depends, as a rule, upon inflammatory action, and is only now and then due to neoplasm. This view of the case has not led to the introduction of any new remedies for consumption, but it has enabled us more definitely to establish indications for remedies already long in use, so that by their methodical application, better results have been attained than were formerly gained at a time when consumption and cancer were regarded as equally incurable, and were somewhat similarly treated.

*Prophylaxis* against consumption requires, in the first place, that, when an individual shows signs of defective nutrition and a feeble constitution, especially if already he have given positive evidence of unusual delicacy, with a tendency to diseases which result in caseous products, *he should be placed, if possible, under influences calculated to invigorate the constitution, and to extinguish such morbid tendency.*

Delicate children, especially such as are born of consumptive or otherwise decrepit parents, should not be suckled by their own mothers; still less ought they to be reared artificially on "pap," but should be confided to good wet-nurses. After weaning the child, let its diet consist almost exclusively of cow's milk, instead of the customary pap of meal or bread, and after it has done teething let it eat a little meat. This diet must be kept up throughout the whole period of childhood, whenever there is any indication of glandular enlargement, moist cutaneous eruption, or any other so-called scrofulous affection, or even when they merely give evidence of a so-called scrofulous habit. It is better to prescribe the exact amount of milk the child must take (after drinking which it may eat what bread, potatoes, or the like, it pleases), than merely to warn the parents in general terms against the immoderate use of bread and potatoes. When the child has drunk milk enough, the other food will do no harm. The common direction, that a "child shall not eat dry food," is wrong. It is better that it should chew and eat its bread dry, so that the amylum which it contains may be properly combined with saliva, whereby it is more thoroughly converted into sugar and is easier of assimilation. Besides this, however, it will drink all the more milk if it eat its bread plain. A similar plan of treatment is of course proper for children, who, instead of inheriting, have acquired a feebleness of constitution which often shows itself at an early date in the form of scrofula, and occasions a predisposition to consumption.

A proper supply of fresh air is of equal importance with regulation of the diet. The facts adduced above, illustrating the baneful effect of continual in-door life in producing scrofula and consumption, are not sufficiently taken into account by many physicians. They very often suffer delicate, sickly children to sit day after day and six hours at a time upon the benches of a crowded school-room, after which they have their tasks at home to prepare, private lessons to take, the piano to play, etc. Cod-liver oil and an occasional month at a watering-place cannot possibly repair the injurious effects of such a mode of life. As soon as the influence of this immoderate "schooling" begins to "tell," a reduction of it, or even a total cessation of it, should be imperatively insisted on. Obstinate opposition to such demands will be often met with, but, in a series of instances in which I have obtained a complete and prolonged respite from education, and made the children spend most of their time in the open air, I have obtained effects at which I was myself astonished, and which completely satisfied their parents that results fully outweighed the serious sacrifices which they had made. People in easy circumstances, who have delicate and scrofulous children, especially if subject to croup and bronchitis, should be induced to spend their winters in the South, so that the children may also pass those months in the open air, which in our climate would be too cold. This is a very common practice in Russia, where the pernicious effects of in-door life during the long winter are very conspicuous.

In adults, when the signs of delicacy and weakness, combined with deterioration of the blood, appear, the use of ferruginous preparations is to be recommended, particularly the chalybeate springs of Pyrmont, Driburg, Imnau, etc. I think that this treatment deserves a more general adoption, as a prophylactic measure against consumption, than it has received hitherto.

Prophylactic treatment of consumption further demands a careful avoidance of all agents calculated to cause hyperæmia of the lungs and bronchial catarrh, and which we have enumerated as exciting causes of phthisis. Persons in whom a tendency to consumption is suspected should be strictly forbidden to inhale an atmosphere charged with smoke or dust, or which is too hot or too cold, as well as to make great efforts in running, singing, dancing, or to drink hot or spirituous beverages. Chilling of the skin is to be guarded against with the utmost care, and the patient should be made to wear flannel next the skin. What we have already said regarding the prophylaxis against pulmonary hypæremia and bronchial catarrh is equally applicable in the present instance.

Finally, whenever there is the slightest suspicion of a predisposition to consumption, every catarrh, no matter how slight, is to be treated with the utmost care, which is not to be relaxed until the catarrh is entirely well. This rule, so obvious from our point of view, is very frequently violated. Many patients fall a victim to the deeply-rooted prejudice, that a neglected catarrh never leads to consumption.

The rules which we have laid down for the prevention of phthisis must be carried out with equal strictness, whether the disease have merely just commenced, or whether it already have made some progress. It is, therefore, superfluous to make separate mention of the indications derived from the cause, as they are identical with those of prophylaxis.

When the air-vesicles of the lung become involved in the bronchial catarrh, the indicatio morbi calls for the usual remedies applicable to chronic inflammation. Above all, the affected lung, like any other inflamed organ, is to be shielded from the action of any new irritation. It is incredible how much this simple rule (so obvious where the nature of phthisis is rightly understood) is disregarded by many physicians. It is a matter of daily occurrence that patients from the better class, suffering from advanced consumption, are not sufficiently urged by their physician to withdraw from their occupation, to throw up their position at the counting-house or office, and to keep away from club-rooms, with their over-heated and tobacco-laden atmosphere. It is often by exposure to irritants like the above, whose effect is so very injurious to the inflamed lung, that the extension of the inflammatory product is aggravated and made to terminate in disorganization, while, by their careful avoidance, the disease is often promptly arrested and brought to a favorable issue. The beneficial effect obtained in consumption, by protecting the affected lung from further detriment, is still more marked among the poorer classes, who seek aid at the hospitals. Many patients are received in a condition so wretched that a speedy death seems imminent, and yet they leave the institution, in the course of a few weeks or months, in much stronger and better condition, and often with a material increase in weight. Soon, however, they return, seeking readmission, their condition having grown rapidly worse again, owing to inclemency of the weather, and to other noxious influences, to which they have been exposed.

Were it not for the very grave objections already detailed, I should counsel most consumptive patients to keep the house during our Northern winter, and to maintain the utmost uniformity of temperature in their chamber, in order to preserve their lungs from further harm. This dilemma may be obviated by making the patient avoid the Northern winter, by sending him to some place where he can spend the greater part of the day in the open air, without risk of taking cold, or of inhaling a raw, inclement atmosphere. This, in my opinion, is the real benefit derivable from change of climate. When a patient has the means, we should never omit to enjoin upon him to make the sacrifice, but the matter must be made plain to him, so that he may not suppose the air of the place to which he is sent has any special curative power upon his lungs. We need not expect any benefit from a residence in Nizza, Mentone, Pau, Pisa, Algiers, Cairo, or Madeira, unless the patient fully understands that he must take care of himself. Otherwise, it were often better that he remained at home. Acting upon this principle, the patient should be sent during the autumn, and before the harsh winter sets in, to Soden, Badenweiler, Wiesbaden, and, above all, to the lake of Geneva, where he may try the grape-cure, and where he is as well protected as he is at home during the summer. None but very intelligent and prudent persons, who we may be sure will stay at home in bad weather, should be allowed to spend the winter at Nizza, Mentone, Pisa, or Pau. When the patients have the means, it is always better to send them to Algiers, Cairo, or Madeira. The comparative merit of these winter abodes is not as yet positively determined, and the indications for preferring Madeira, Algiers, or Cairo, in particular cases, or for certain stages of the disease, are so indefinite as to be of little value. One principle, however, always obtains: *that the patient, wherever he may be, must live circumspectly, and remain under the charge of an intelligent and strict physician.*

For patients who are unable to seek a milder climate, the use of a "respirator," a wire gauze, warmed by the breath, through which the external air is inhaled, is advisable. A handkerchief held before the mouth, however, which also is soon warmed by the expired air, will answer the same purpose, and, indeed, is really better than a "respirator," as it is not, like the latter, liable to become too warm.

When the invasion of the air-vesicles by acute catarrh, or the rapid spreading of a catarrhal pneumonia, is accompanied by violent symptoms, when high fever sets in, when the sputa become bloody, and the patient complains of lancinating pain upon drawing breath, and upon coughing, local depletion, by means of leeches or cups, and the application of cataplasms, should be resorted to. At the same time, the patient must be required to keep his bed until all symptoms of the acute attack, or of the exacerbation of the old inflammatory disorder of the lung, be past. The fits of shivering, which come on regularly every evening, in many cases of phthisis, and which sometimes actually amount to rigors, have often been observed to cease if the patient remain in bed. And, upon closer observation, it has been found that not only does the chill which heralds the evening access of fever, but all the other febrile symptoms, especially the rise in temperature, undergo marked improvement while the patient remains in bed for a few days. There is nothing strange about this, if, instead of regarding the hectic fever of consumptives as something peculiar, as an *ens sui generis*, we look upon it as a fever due to chronic inflammation. The fever which accompanies bronchial catarrh, pneumonia, or inflammation of any other organ, increases and diminishes as the disease grows better or worse, and it is just the same with the hectic fever of phthisis. Hence, if resting in bed, such as we generally recommend in other inflammatory disorders, have a beneficial effect upon the pneumonia of consumptives, it will tend also to mitigate their fever.

The use of the alkaline muriate mineral waters, which is often so beneficial in simple catarrh, is equally useful in some cases of consumption. According to our view of the disease, this effect (which of course all believers in the theories of *Laennec* will deny) is not more enigmatical than that which these waters produce upon a simple catarrhal inflammation, which does not involve the substance of the lung. The idea, that the use of the waters of Ems and Obersaltzbrunner is contraindicated by the presence of fever, is merely one of the results of imperfect observation. It is not the mineral waters which disagree with the fever, but the journey to the watering-place, and the promenades at the springs. As we have said before, a patient with any appreciable degree of fever ought to be in his room or in his bed.

A continued abode in elevated regions, where, without any apparent reason, consumption is rare, is also advisable for consumptives, when their disease depends upon chronic pneumonia. I fully approve of the customary practice of sending phthisical patients to spend their summer at Heiden, Gais, Weissbad, Kreuth, etc., although I think but little of the "curds and whey treatment" which is practised there.

In tuberculous phthisis, and in secondary tuberculosis, it is out of our power to meet the indications derived from the disease itself.

*Indicatio Symptomatica.*—Fever is the symptom which principally demands treatment, whenever it persists at all severely, in spite of the remedies directed against the main disease. Anti-pyretics very properly play a most important part in the therapeusis of consumption. It is not that these remedies exert any more direct influence upon chronic pneumonia than they do upon croupous pneumonia or typhus, or upon any other of the many maladies in which they are so much prescribed, often, indeed, without any very clear idea as to what is to be expected of them. But, if we know that the discharge of mucus and of pus-cells has but little to do with the exhaustion of the patient (indeed, it is often far more profuse in a simple bronchial catarrh), and that the fever is really his most formidable enemy, it follows, of course, that we must use every means at hand of combating this enemy.

Digitalis and quinia have a well-merited reputation, as means whereby we often succeed in arresting the abnormal calorification, and reducing the animal heat, in spite of the continuation of the disease. Digitalis is the principal ingredient of the much-employed Heim's pill. (℞. pulv. herb. digitalis ℈ ss., pulv. rad. ipecac., pulv. opii puri āā. gr. v., extract helenii q. s. u. f. pil. no. XX. consp. pulv. rad. irid. flor. S. a pill three times daily.)

The addition of a scruple of quinine to the above prescription becomes all the more appropriate, the more periodical the type assumed by the fever, the more severe its evening exacerbations become, and the more pronounced the chills by which they are ushered in. I am so much in the habit of using Heim's pill with or without quinine, in consumption, whenever the fever proves refractory to the other remedies heretofore mentioned, that it has become a very common prescription at my clinic. Now and then, when I am a good deal consulted by phthisical patients, I prescribe it three or four times in one day. At the clinic, exhibition of the pills is suspended whenever a distinct reduction of the temperature and of the frequence of the pulse becomes apparent, and is resumed as soon as the effect subsides. In consultation practice, I have repeatedly found that the patients pretty soon learn to judge for themselves when it is time to stop the pills, and when to resume them.

The subject of antipyretic treatment of consumption may, with great propriety, be immediately followed by that of the diet of phthisical patients, for the same reason which induced us to treat the subjects of fever and emaciation in immediate conjunction. A man who has fever which is rapidly consuming him, stands in far greater need of a supply of nutriment than one who has no fever. The fever of a consumptive patient often lasts for months, so that the danger that it will wear him out is greater in his case than in one of acute febrile disease of brief duration. Hence it follows that phthisical patients require the richest possible diet which will agree with them. It is often said, but without any proof whatever, that food excites the fever, and (independently of the English practice) even here (in Germany) we only keep a patient on fever-diet—that is, we only deprive him of nourishing food until it becomes evidently dangerous to persist in so doing. As soon as this is evident, the so-called law of nutrition is utterly ignored, or, rather, it is flagrantly violated. In selecting suitable nourishment for consumptives, articles commended, time out of mind, by rude experience, are found to be in complete agreement with the current physiological laws of assimilation and nutrition. All the food which is regarded as especially proper for phthisical patients contains large quantities of fat or of fat-generating matter, and a comparatively small portion of protein substances. This accords with our experience, that the production of urea, and hence the destructive assimilation of nitrogenous constituents, is augmented by an increase of the supply of protein substances, while, by a simultaneous free supply of fat or fattening food, the destructive assimilation and consumption of the organs of most importance in the body are diminished. Thus the use of milk, to which little children owe the plumpness of their limbs, and from which corpulent persons do well to abstain, cannot be sufficiently urged upon consumptive persons. It is altogether useless, however, and indeed wrong, to remove the casein of the milk, and to give it in the form of whey, unless, indeed, the whey agree with the patient better than the milk, which is rarely the case. I often order my patients to drink a pint of milk "warm from the cow," three times a day, but have no other object in so doing than that of preventing the milk from being skimmed, which is impossible immediately after milking. The milk of animals which pasture in the mountains, such as goat's milk, but, above all, ass's milk, is in especial repute, and it is desirable to send patients, who can travel without danger, to places where there are dairies where a supply of good fresh milk is to be obtained. Where this cannot be done, the "milk-cure" must be practised at home. The name is of importance, in order that the patients may have faith in the treatment, and follow it out punctually. I have treated a great number of patients who, as soon as they found that they increased appreciably in weight, for half a year at a time drank three or four pints of milk daily without repugnance.

The use of cod-liver oil is also highly commendable, and, when it agrees well with the patient, may be combined with plenty of milk. It is more than doubtful whether this oil, which is hardly ever withheld in phthisis, at all events in Germany, exerts any specific influence upon the disease. The quantity of iodine in it is so trifling, that it cannot be taken into account, hence it is probable that all its beneficial effects are solely due to the large amount of fat which it affords. This is all the more likely, as *dog's fat* is a popular remedy for consumption, as ancient and well-tried as cod-liver oil.

Of late years I have obtained very good effects from an extract of malt, prepared by *Trommer*. This preparation of *Trommer* is not a strong beer, containing a large amount of alcohol and carbonic acid, like the *Hoff's malt extract* so greatly extolled, but is a genuine extract resembling other officinal extracts, and consists of the soluble constituents of the malt, and of the bitter extractive matter of the hops, and can be prepared by every apothecary. One hundred parts of it contain about seventy-six parts of grape sugar, or malt sugar, dextrin, bitter of hops, resin of hops, and tannin, seven parts of albuminous or protein substance, eighty-two hundredths of a part of phosphate of lime and magnesia, eighteen hundredths of alkaline salts, and sixteen parts of water. The patients almost always enjoy two or three tablespoonsful of it daily, and it usually agrees well with them. It may be diluted in spring-water, mineral water, or warm milk or other liquid.

Broth, made of coarsely-broken rye-meal, which contains a good deal of gluten, besides the amylum, is a good food for consumptives, and has long enjoyed such a reputation. Soup of lentils and bean-meal (revalenta arabica), as well as the various preparations of chocolate, mixed with cacao-meal, and sold under various names, is also appropriate.

Jellies of animal or vegetable substance are much less desirable, such as the snail-soup, and the jelly from the Iceland moss.

With respect to the symptomatic treatment of the cough and expectoration, we simply refer to what has already been said regarding the treatment of bronchial catarrh. An indiscriminate use, one after another, of the so-called expectorants is as absurd in the treatment of the chronic bronchial catarrh which accompanies phthisis as it is in any other form of catarrh. The sweet, mucilaginous, "soothing," demulcent articles are least serviceable of all. Precisely according to the conditions laid down above, the alkaline chlorides may be required at one time, at another senega, squills, or other stimulants may be indicated, and at still another the articles which diminish secretion. As we have already expressed our preference for the balsams and resins for the latter purpose, I must again say a word or two in favor of the saccharum myrrhæ, and of Griffith's mixture, adding, however, that acetate of lead is held in great esteem by many authorities as a remedy for the condition in question. (In almost every case where acetate of lead is used it is given in combination with opium, to which some of the effect attributed to the lead is certainly due.)

The narcotics are to be employed in order to allay the cough, and are quite indispensable in consumption. As we have said already, it is not the soothing, soporific action of the first few doses of the opium or morphine which gratifies the patients, but it is because they find that they cough less and more easily, "that their cough is looser;"

and, indeed, when we consider that coughing is an irritant to the bronchial mucous membrane, which is the principal source of the secretion, it seems quite probable that a diminution of the inclination to cough may result in a decrease of the expectoration. Nevertheless, it is best not to commence using the narcotics too soon, and, instead of opium, we should begin with small doses of something else, as, extract of lactucaria virosa, gr. ss to gr. j, in powder, or in the form of a syrup. By a too early resort to narcotics, it may happen that they fail of effect at a later period, when the need for them has become most urgent, as when the tormenting cough of a laryngeal phthisis deprives the patient of rest both by night and by day. It seems also, that, as soon as it becomes necessary to give large doses of opium, the progress of the consumption becomes more rapid, an additional reason against a too hasty employment of a remedy which becomes indispensable to the patient.

For the night-sweats we may order small doses of "Haller's acid," or the patient may drink a cup of cold sage-tea, if the antipyretic treatment fails to do good. The efficacy of the above articles is somewhat questionable, no doubt, but it would be cruel to tell the patient that there are no means of relief from this distressing symptom. Some physicians recommend the boletus laricis (a very variable article), as a most efficient remedy against the night-sweats of consumption.

With regard to the treatment required by the complication of laryngeal and intestinal phthisis with consumption of the lungs, as well as that demanded by the secondary diseases of the liver and kidneys, etc., we must refer to the sections in which affections of those organs are described.

## CHAPTER XIV.

### ACUTE MILIARY TUBERCULOSIS.

**Etiology.**—Acute miliary tuberculosis, which is not to be confounded with acute ("galloping") consumption, depends upon an eruption of tubercles in the lungs as well as in most other organs, and is accompanied by the symptoms of an acute disease. In the great majority of cases the disease is seen in persons whose lungs or other organs contain old caseous deposits. This fact, and the cir- [illegible] that the symptoms and course of acute miliary tuberculosis [illegible] resemblance to those of the acute infectious diseases, ppear highly probable that the malady arose from in- d by the caseous products (*Buhl*), were it not that h rare occurrence of the disorder, unpreceded by caseous deposit, contradicts this plausible hypothesis. We must, therefore, content ourselves by stating that, in most cases, acute miliary tuberculosis is a secondary disease, arising, in some manner as yet unknown to us, from the pernicious effect of the cheesy deposit, but that may also proceed from other causes, of whose nature we are equally ignorant.

Anatomical Appearances.—If we find, upon dissection, that the lungs are studded uniformly from top to bottom with miliary tubercles, if the miliary nodules present that gray, translucent appearance of fresh tubercle, if the surfaces of the pleura be also strewed with miliary tubercles, we may decide with positive certainty that the patient has had acute miliary tuberculosis, even though we know nothing of what the course of the disease has been. In chronic tuberculosis this uniform dissemination of the tubercle is never found, and yellow, caseous granulations always coexist with the new gray tubercles, showing that the deposit has been a gradual one. In most cases of the acute disease, the peritonæum, the liver, the spleen, the kidneys are covered by miliary tubercles. Finally, especially in young persons, numerous granulations are often found in the pia mater, particularly at the base of the brain, about the pons, and the chiasm of the optic nerves, together with acute hydrocephalus of the ventricles.

The parenchyma looks injected and more or less infiltrated with serum, otherwise it is generally free from inflammatory or other nutritive disturbance, with the exception of the traces of former disease which may be there. The corpse of a person who has died of acute miliary tuberculosis resembles that of one who has died of an acute febrile disease, the resemblance commencing during life and continuing after death. The blood is dark and liquid, and settles to the most dependent points, giving rise to extensive pulmonary hypostasis. The muscles are red, and even the spleen is often somewhat swollen and softened.

Symptoms and Course.—When an acute miliary tuberculosis develops at an advanced stage of consumption, complicated with hectic and night-sweats, it is very difficult of recognition, inasmuch as it can hardly be decided whether the fever and the rapid decline of the patient are due to the original complaint or to the complication. Physical examination of the chest gives negative information as to the new deposit of miliary tubercles; as the innumerable little granules, being everywhere enclosed in tissue containing air, do not modify either the sound upon percussion or the respiratory murmur, although the disproportion between the intense dyspnœa and the trifling extension of some old point of induration perhaps may aid the diagnosis.

The disease assumes a different guise when it attacks persons in good health, or those whose chronic pulmonary affection has hitherto escaped attention. It then not unfrequently begins with repeated rigors, great frequence of the pulse, and severe constitutional disturbance, symptoms often hard to interpret, as they are attended by no tokens of local disorder. The frequence of the pulse often becomes exceedingly great, abundant sweats set in, the patient sinks visibly from day to day, the tongue becomes dry, the sensorium deranged, he becomes delirious or lies supine in a state of stupor. A rapidly-increasing prostration, cough, and dyspnœa accompany these symptoms, it is true, but the most persistent physical examination of the chest reveals nowhere that the substance of the lung is infiltrated. No sounds can be perceived, save a few fine rhonchi and scanty *râles*. The symptoms which we have depicted are so very like those of typhus, that the most experienced diagnosticians acknowledge to having met with instances in which a diagnosis between the two was absolutely impossible, and where patients dying with a diagnosis of typhus really had died of tuberculosis, and conversely. The less violent the symptoms of catarrh in acute miliary tuberculosis, the smaller the clew afforded by the spleen, the more rapid the march of the malady, so much the more difficult does the distinction become. The patient may succumb to miliary tuberculosis, after the lapse of a fortnight, or a few days longer, or about in the same time in which patients usually die of typhus. More rarely death does not take place until the end of the fifth or sixth week. The patient perishes, as we have said, consumed by fever, just as he falls a prey to fever, too, as a rule, when he dies of typhus. The pulse becomes smaller and more and more frequent; finally, the pulmonary veins are no longer able to pour their blood into the imperfectly-emptied heart, and œdema of the lungs, palsy of the bronchi, and suffocative effusion are established. If tuberculous basilar meningitis accompany the attack, its course is modified (see appropriate chapter) and the fatal termination takes place with even still greater rapidity.

DIAGNOSIS.—At the outset of the disease, if the chills recur with some degree of regularity, it may be mistaken for intermittent fever. We shall soon observe, however, that the intermissions are not complete, that quinine fails in its effect, that the complaint is attended by a disturbance in the respiratory function, which is unusual in intermittent; that the frequence of the pulse is constantly on the increase, and that the entire character of the complaint is more pernicious than that of simple intermitting fever.

In other cases the disease, at its commencement, resembles an extensive bronchial catarrh, accompanied by fever, especially if the cough be very violent and distressing; but here, too, all difficulty of distinction soon vanishes, as the violence of the fever, the rapid collapse, and the malignant course of the malady, but especially the shortness of breath which often renders it impossible for the patient to breathe in a recumbent position, and which is in striking contrast with the absence of physical signs of disease, afford data for diagnosis.

A differential diagnosis between miliary tuberculosis and typhus is based upon the following points:

1. In tuberculosis, the cough and dyspnœa appear, as a rule, at an earlier period and with far greater intensity than in typhus. In exanthematic typhus, it is true, we likewise find early and violent bronchitic symptoms; but here the distinction is easy, as the eruption of exanthematic typhus is highly characteristic and scarcely to be overlooked, while there is no eruption in acute miliary tuberculosis.

2. In abdominal typhus (typhoid), likewise, we rarely fail, after careful and repeated search, to discover a few spots of roseola upon the upper region of the abdomen, which do not exist in acute miliary tuberculosis.

3. Enlargement of the spleen can rarely be found in acute miliary tuberculosis, and when found scarcely ever is an enlargement of much magnitude, while we hardly ever fail to find it in abdominal typhus: and even though it were not found, in exanthematous typhus the existence of the eruption would render this clew almost unnecessary.

4. Meteorism, liquid stools, tenderness in the ileo-cœcal region, are seldom absent in abdominal typhus. These symptoms are not observed in acute miliary tuberculosis.

5. Typhus rarely supervenes upon chronic disease of the lungs, while acute miliary tuberculosis seldom attacks any save those who are suffering from such disease. Dulness at the apex of either lung is therefore of great diagnostic significance.

6. *Wunderlich* has observed that the temperature in acute miliary tuberculosis is much lower than in typhus, seldom reaching 104° F., and is out of all proportion to the enormous rapidity of the pulse.

PROGNOSIS.—Prognosis as to the issue of acute miliary tuberculosis must be almost absolutely unfavorable. Only a very few observations (*Wunderlich*) allow us to suppose that tubercles thus deposited may become atrophied, and the malady terminate in recovery. The cases, too, in which the acute disease has become arrested, and chronic tuberculosis and phthisis have followed, certainly must be considered as among the greatest of rarities. The more violent the fever, the more pronounced the brain-symptoms, so much the sooner is the end to be expected.

TREATMENT.—The treatment of acute miliary tuberculosis is of course a mere treatment of symptoms. The most important symptom is the fever; for it is of the fever alone that the majority of those attacked perish. Large doses of quinine should be given, particularly at the outset of the disease and as long as the rigors continue to occur, and at a later period use digitalis, nitre, and the acids. Little success, however, is to be anticipated. For the dyspnœa, cold is to be applied. Combat the cough with narcotics; and, should appearances lead us to suspect the existence of meningeal tuberculosis, apply ice to the head.

## CHAPTER XV.

### CANCER OF THE LUNG.

Etiology.—The pathogeny and etiology of this malady are as obscure as those of the malignant neoplasms in general.

Cancer of the lung is a somewhat rare disease, and primary cancer of this organ is of especially unusual occurrence; that is to say, the substance of the lung is scarcely ever the point at which the first traces of it develop themselves. Cancer of other organs, particularly of the breast, almost always precedes cancer of the lungs.

Anatomical Appearances.—In the lung, cancer assumes almost exclusively the medullary form, far more rarely that of the scirrhus or of alveolar degeneration. It sometimes assumes the form of rounded isolated masses, varying from the size of a hemp-seed to that of a fist, constituting cancerous nodules of a marrowy appearance and soft consistence, which, when they touch the pleura, are apt to show a flattened or umbilicated depression. Sometimes the disease appears as the so-called infiltrated cancer. Unlike the previous variety, the latter form does not present a distinct limit between the cancer and the surrounding parenchyma, but makes a gradual transition; nor does the disease present the rounded contour of cancerous nodules.

The old hypothesis, that, in the latter case, we had to do with a conversion of an infiltration into cancer, has been abandoned; and it is now believed that, in the origin of infiltrated cancer, after the transformation into cancer-cells of a few of the connective tissue-cells of the matrix of the lung, and of a few of the epithelial cells of the vesicles, this conversion is propagated into the neighboring connective tissue, and into the connective tissue-cells of the adjacent alveoli. On the other hand, with regard to the appearance of isolated cancerous nodules in the lung, we must suppose that here, too, the cancer-cells originate from the elements of the tissue, and then proliferate without further implication of the contiguous tissues in the disease. The enlargement of the tumor, therefore, is due to proliferation of the original cancer-cell alone; the surrounding pulmonary substance being pushed aside and compressed.

It is exceedingly rare for medullary fungus of the lung to soften, and break down, so as to form cavities. The disease is much more liable to extend into the pleura, and, as the pleural folds rapidly adhere, to spread through them into the walls of the chest, which it often penetrates.

SYMPTOMS AND COURSE.—In the great majority of instances, no characteristic marks of cancer of the lung are to be observed, and it is hardly ever possible to prove the existence of the disease with certainty, except in cases wherein a carcinomatous breast has been extirpated, or in which extensive cancerous disease of other parts of the body can be discovered. Should dyspnœa, cough, blood-spitting, and pain in the chest, symptoms indicative of chronic disease of the lung, appear in such a case, instead of apprehending the formation of tubercle, we should bear in mind the rarity of tuberculosis in cancerous persons, and of the frequent relapses of the malady, in the form of pulmonary carcinoma, after extirpation of cancerous masses.

Diagnosis will be confirmed if percussion and auscultation show a consolidation of the substance of the lung, especially as, unlike tubercle, cancer is not habitually situated at the summit of the lungs.

We are very seldom able to prove the existence of any characteristic objects in the sputa. The diagnosis is more commonly rendered certain by the perforation of the thorax by the disease and its extension into the integument.

TREATMENT.—Of course, there can be no idea of treating a cancer of the lung. The hyperæmia in its adjacent parts, the œdema, the hemoptysis, must be treated according to directions already given.

# SECTION IV.

## *DISEASES OF THE PLEURA.*

## CHAPTER I.

### INFLAMMATION OF THE PLEURA—PLEURITIS, PLEURISY.

Etiology.—As we shall find presently, there are two forms of pleurisy. The first form merely causes thickening of the pleura, and adhesion of its opposing surfaces. The second also produces thickening, but at the same time gives rise to an effusion into the pleural sac, containing more or less of fibrin and of young cells. The thickening and adhesion of the pleural surfaces are due to proliferation of the normal connective tissue of the pleura. The pleuritic effusion is the result of an interstitial exudation. The young cells, which the effusion contains, owe their origin to a proliferation of the connective tissue corpuscles of the pleura, and of the epithelial cells which cover its surface.

Regarding the essential points in the etiology of pleurisy, we may refer to what has already been said with regard to the etiology of pneumonia.

We must here denounce the impropriety of calling all cases of pleurisy secondary pleurisy, which, instead of attacking robust and vigorous persons, occur in subjects with broken-down constitution, or in individuals who have already suffered from some other disease. Even the pleurisy which so often occurs in Bright's disease is not, in my opinion, a secondary disease, dependent upon the renal affection, but should rather be looked upon as a complication. The frequence of such complications, and the especially common occurrence of pleuritis in debilitated and depraved constitutions, and among convalescents after protracted disease, depend upon the increased predisposition, which such individuals possess, for all kinds of inflammatory diseases, and especially for the one in question. A very trifling exciting cause is requisite in this class of persons to provoke the malady; but it never arises without provocation of some kind.

The case is different in the pleurisy which sometimes accompanies septicæmia, puerperal fever, scarlet fever, and other infectious disorders. Such a pleurisy arises independently of the action of any new irritant, and forms one of the nutritive derangements which proceed from infection of the organism by putrid matter, scarlatinous poison, or the like. This *secondary* pleurisy, which generally produces a puruloid exudation full of young cells, is attended by inflammation of other serous membranes.

The exciting causes of pleurisy are—

1. Injuries of the ribs and pleura, and the entrance into the latter of foreign bodies, such as pus, blood, air, and the contents of cavities. Such exciting causes generally give rise to a form of pleurisy, accompanied by a very profuse sero-fibrinous exudation into the pleural sac.

2. Pleuritis often arises through propagation of inflammation from neighboring organs, as from the lungs to the substance of the pleura. In these cases the exudation generally is scanty and fibrinous, although it sometimes is very copious and sero-fibrinous.

3. Next in order come the very numerous instances in which pleurisy is caused by the advance to the pleura of neoplasms, especially of tubercle and carcinoma. Here the pleurisy is dry, or else it results in adhesions of the opposing surfaces of the pleura, or else a more or less plentiful effusion into the sac may form, or, finally, tubercle or cancer may develop in the pseudo-membrane.

4. Pleurisy is often the consequence of exposure to cold, or to the action of other atmospheric or telluric influences, of which we have no definite knowledge. In this form, which is an independent, idiopathic disease, and which is usually called *rheumatic pleurisy*, there is a great deal of variety as to the quantity and character of the effusion.

ANATOMICAL APPEARANCES.—In commencing pleuritis, the pleura is reddened by injection proceeding from the sub-serous connective tissue, and producing fine rose-red points and stripes upon its surface. Besides this distention of the capillaries, we often find slight extravasations of blood, ecchymoses, forming irregular dark spots, in which the ramifications of small vessels are visible. The tissue of the pleura is infiltrated, the epithelium is nearly all cast off, the surface, formerly smooth and glossy, looks dull, the pleura itself is somewhat swollen. Gradually the free surface begins to assume a rough, shaggy appearance. This is due to the development of minute delicate folds, and papillary granulations, which are firmly attached to the surface, and are not to be confounded with fibrinous deposits. Microscopically, these granulations consist of newly-formed fusiform cells, and tender filaments of wavy connective tissue, with considerably-elongated capillaries, which are coiled into loops within them (*Foerster*).

These changes occur in every form of pleuritis, whether effusion take place in the pleural cavity or not, whether the latter be profuse or scanty, contain much or little fibrin, or many or few pus-corpuscles. It is to this source alone that pseudo-membranes and adhesions of the pleura owe their origin.

The most common forms of pleurisy are—

1. *That in which no symptoms occur*, excepting those just described, and to which we may give the name of pleuritis sicca, dry pleurisy, or pleurisy with purely nutritive exudation. It is true, that we but rarely have opportunity to make anatomical examination of a dry pleurisy in its earliest stage. However, whenever this has been possible (*Foerster*), no free exudation has been found to exist, the outgrowths from the pleura just described forming the sole abnormity. Besides this, however, very extensive adhesions of the pleura are often found, which have formed almost without giving rise to any symptoms, and this fact would indicate that they must occur without exudation, for we find that very small exudations are accompanied by very great pain.

2. *Pleurisy with scanty, but very fibrinous, exudation.* Such a pleurisy we almost always see accompanying croupous pneumonia or complicating chronic affections of the lungs. It may also occur as an independent disease. Here the inflamed pleura, having undergone the alterations above described, soon becomes coated by an extremely delicate membranous coagulum of fibrin, which causes it to appear more opaque, so that we cannot discern the injection, or ecchymosis of the pleura itself, until we have scraped off the fibrin with the scalpel-handle. In other cases, this very fibrinous effusion is somewhat more profuse, and we may then observe upon the pleura a white deposit, half a line or more in thickness, somewhat soft, and very much like a croup membrane. Of course, the exudation in these cases was originally liquid, and only coagulates at a later period; nevertheless, we are often unable to find any liquid effusion besides the coagulated one in the cavity of the pleura. When this form of pleuritis recovers, the fibrinous deposit, after undergoing fatty degeneration and liquefaction, is absorbed, the outgrowths of the opposing surfaces of the pleura are brought into contact, and adhesions generally ensue.

3. *Pleurisy with abundant sero-fibrinous exudation.* The alterations in the tissues of the pleura are usually very extensive in this form of the disease, both upon the pulmonary and costal surfaces; but, in addition to this, an effusion of serum takes place in the pleural sac, amounting, not unfrequently, to two or three, and, indeed, even to ten pounds or more. This exudation consists of two components—a yellowish-green serum, and a quantity of coagulated fibrinous masses.

Part of the latter floats in the serum in the form of flakes and lumps, another part traverses the serum in the form of a loose net-work, while a third portion is precipitated upon the pleura, upon which it lies in the form of a membrane. The longer the effusion remains, so much the stronger and more rigid do the masses become, until they finally grow fibrous, without, however, taking on any organization. Both in the serum and in the fibrinous deposit we find a few pus-corpuscles, so that the transition from this form of pleurisy to the next, in which pus-corpuscles are far more abundant, is quite gradual. The greater the quantity of pus, so much the more turbid is the serum, and the more yellow the deposit. The proportion between the serum and the fibrin varies, although here, too, we are not warranted in regarding fibrinous exudation as the consequence of a hyperinosis (augmentation of fibrin in the blood). Indeed, according to the old-fashioned theory, it is far more probable that a pleurisy, in which a great amount of fibrin is secreted in the pleura, also causes the increased quantity of fibrin in the blood. The exudation often seems to receive accessions, and to increase by fits and starts. As these after-flows do not come immediately from the vessels of the pleura, but from the thin-walled vessels of the young connective tissue, we often find an admixture of blood in the serous effusion of chronic pleuritis, in consequence of rupture of the delicate capillary walls, thus forming pleurisy with hæmorrhagic exudation. We constantly find agglutinations of the opposing surfaces by fibrinous exudation, as well as commencing adhesions, surrounding the effusion, whereby the latter is often incapsulated. This is a condition of great importance in the symptomatology of the disease.

According to the lucid and concise account of *Rokitansky*, the changes which take place in the thorax and its contents, in consequence of extensive effusion, are as follows: "The thorax is dilated in a manner more or less apparent, the intercostal spaces are widened and prominent, the diaphragm is forced down into the abdomen, the mediastinum and heart are displaced to the other side, or, when the effusion is symmetrical, lie in the middle of the chest. The lung itself is compressed to a degree corresponding to the amount of the effusion, and, unless old adhesions offer resistance, it is constantly pushed upward and inward against the mediastinum and back-bone. We find it reduced to the fourth, sixth, and even to the eighth part of its normal volume, and flattened into a cake, its color is pale reddish or bluish gray, or lead color, and its consistence is leathery, tough, and void of blood and air, and in a state of atrophy at the edges and surface. It is coated externally by the coagulum of fibrin, which extends from the costal to the pulmonary pleura. In partial pleuritis, the displacement and compression are limited to a portion of lung corresponding to its seat and extent."

The lung upon the unaffected side is always the seat of intense collateral fluxion, and, in fatal cases, of collateral œdema. Should recovery take place in this form of pleuritis, the exudation gradually becomes more and more concentrated (so that the absorption proceeds at first far more rapidly than it afterward does). The liquid portion may at length disappear completely; the pleural surfaces, roughened by fibrinous deposit, coming into contact. The fibrin also undergoes fatty metamorphosis, liquefies, and is absorbed, and then an adhesion of the pleural surfaces, which are usually much thickened, always takes place. Sometimes yellow, cheesy masses, consisting of remnants of unabsorbed fibrinous deposit and cellular elements of the exudation, are found imbedded between the adhesions.

When absorption takes place early, the compressed lung may again become pervious to the air, and may expand; the intercostal spaces may return to their normal state, and the mediastinum, diaphragm, and the dislocated heart and liver, may all regain their proper places.

In other cases the alveoli become agglutinated or adherent by continued pressure, or else dense fibrinous deposits upon the compressed lung prevent its reinflation. The time required for the production of this condition cannot be given with accuracy. If absorption of the exudation should afterward take place, a vacuum tends to form, to fill up which, the thoracic wall and the adjacent organs suffer displacement. The affected side of the chest sinks in, and may present a concave instead of a convex surface; the intercostal spaces become narrower, until the ribs finally touch; the shoulder sinks, and even the spinal column becomes curved. In pleuritis of the right side, the liver, previously deeply depressed, is now dislocated far in the opposite direction, sometimes as high as the third rib. In pleurisy of the left side, the heart, at first often displaced to beyond the right edge of the sternum, now is drawn back as far as the left axillary line.

4. *Pleuritis with purulent effusion. Empyema, Pyothorax.*—The liquid part of the effusion is here so rich in pus-corpuscles as to form an opaque, yellow, thick fluid. The fibrinous portion also contains great quantities of pus-cells and seems soft, and of a very yellow color. Here too, the exudation, and not only the serous part of it, but the fibrin and pus, after undergoing the often-mentioned metamorphosis, may be absorbed; but there is another sequel to pleuritis sometimes, and it most frequently follows this form of the malady. Not only are pus-corpuscles generated upon the free surface, but they are also formed within the tissue of the pleura itself. The latter becomes opaque and softens, and irregular losses of substance occur. Should

they be situated upon the costal pleura and penetrate deeply, external perforation of the empyema may take place, and in fortunate cases, especially if the lung remain capable of redistention, recovery may be the result. In similar manner, a penetration of the empyema into the lung and its discharge by way of the bronchi sometimes happen, but a recovery in such instances is rare.

SYMPTOMS AND COURSE.—Dry pleurisy has no symptoms, or, at least, if it have symptoms, they cannot be distinguished from those of the disease which it accompanies. We sometimes find adhesion of the entire pleural surface in the bodies of persons who never have been seriously ill. Extensive and rigid adhesions of the pulmonary and costal pleuræ hinder the two surfaces from sliding upon one another, and thus prevent a uniform expansion of the lung during inspiration (see vicarious emphysema). The consequence often is a slight dyspnœa, which is only felt, when unusual bodily exertion or other cause excites a demand for an increased supply of oxygen.

*Pleurisy with scanty fibrinous exudation* is accompanied by severe piercing pain when a breath is drawn; the suffering produced by the limited and slow movement of the pleura, during ordinary breathing, is far greater than that arising from the strong and rapid motion of forced respiration. Coughing and sneezing are especially painful to the patient, as these acts compress the inflamed pleura from within. In like manner a pressure upon the ribs and intercostal muscles affects the pleura immediately, and greatly increases the pain. The respiration of the patient is shallow and cautious. The body is generally bent toward the affected side, as this attitude lessens the tension of the intercostal muscles and its inflamed covering. Besides the pain, some patients have a distinct sensation of friction, or of scratching at some point of the thorax. There is also cough, as a rule; although cases now and then are observed where there is absolutely no cough, and it has not as yet been determined satisfactorily whether the cough is a result of reflex action from the inflammatory irritation of the pleura, similar to that arising from irritation of the bronchial mucous membrane, or whether it is due to a complication of pneumonia or bronchitis with the pleurisy. The pleurisy with scanty fibrinous exudation, unless accompanied by extensive and severe inflammation of the lung, is usually unattended by fever, or other serious derangement of the health. Many patients never even keep their room, and often go on foot to the clinic, or to the office of their physician, for medical aid.

We have already stated that the pleuritic stitch, which is one of the most painful symptoms of croupous pneumonia, and which indubitably owes its origin to the almost constant complication of the latter disease with the form of pleuritis now under consideration, is generally of briefer duration than the other symptoms of pneumonia. Perhaps this is because the pleural surfaces cease sliding upon one another, where a large portion of the lung has become infiltrated. But even when the disease occurs spontaneously, or when it supervenes in chronic disease of the lung, the pain usually ceases in a few days, especially if properly treated. Its persistence for weeks is an exceptional occurrence, and should cause suspicion of grave disease of the lung.

*Pleurisy with profuse sero-fibrinous exudation* sets in quite often, with violent general phenomena, and severe symptoms of local disease, in a manner very like the commencement of pneumonia. The malady begins acutely, and runs an acute course. Ushered in by a severe rigor, it is followed by intense fever, with the full and frequent pulse, the headache, and pain in the back and limbs, the coated tongue, and the parching thirst, which we see in almost all violent inflammatory diseases. There may, however, be more than one chill, and there are often several, the succession of which may take on so well-marked a tertian type, that it is quite possible to mistake an incipient pleurisy for an intermitting fever. A sharp pain, usually referred to the side of the chest, is also felt at the beginning of this variety of pleurisy, into which the form last described often passes, the exudation becoming more copious and richer in serum. As the disease advances, the pain abates somewhat, and often ceases altogether, before the pleurisy has attained its climax, or, especially before the effusion is complete. The cough, which scarcely ever fails, and which is often extremely distressing and persistent, is sometimes plainly attributable to the collateral hyperæmia, and collateral œdema of the uncompressed part of the lung. At other times its source is obscure. Besides these symptoms, there is dyspnœa, which becomes aggravated as the effusion increases, and which often becomes extremely severe. It is important to bear in mind the fact that a part only of the dyspnœa is caused by pressure of the effusion upon a portion of the lung, and that the collateral hyperæmia and œdema arising in the uncompressed portion, and by which the breathing surface of the latter is materially diminished, play an important part in the production of dyspnœa. At all events, even where the effusion is very large, the difficulty of breathing diminishes, and often ceases altogether, just as it does in croupous pneumonia, as soon as the fever abates, and with it the need of additional oxygen.

After increasing in intensity for six or eight days, a sudden improvement may take place, just as in croupous pneumonia, the general disturbance and dyspnœa undergoing a marked decrease, or even ceasing totally within a few hours. This depends upon a rapid abatement of the fever. In fortunate cases the reabsorption of the effusion also begins immediately and progresses rapidly. As already stated, the absorption goes on most rapidly at the outset, and, as the volume of the liquid decreases, and its concentration becomes greater, absorption grows slower and slower, so that, even weeks after the patient has apparently entirely recovered, a remnant of the exudation can still be found.

Next to these cases, which are acute from beginning to end, come those which, being acute at the outset, afterward take on a slow and tedious character. The fever moderates at the end of the first week, or a little later. The exudation makes no further progress; but we wait in vain for a complete subsidence of the febrile general disturbance, and for absorption. At last the exudation begins to diminish; the air once more enters the compressed parts of the lung; but, in the midst of this apparently favorable prospect, we again one day find the patient short of breath, coughing hard, anew spitting bloody froth. The fever, too, has grown worse; and, if we now examine the chest, we find that the effusion has increased by a hand's breadth, and extends higher than ever. In this way the disease, originally acute, drags on with fluctuating symptoms for months, and, as a rule, terminates fatally.

Thirdly and lastly, there are a great many patients in whom this form of pleuritis develops slowly, and often without attracting attention, its subsequent progress being of an equally tedious character. There is no inflammatory fever, and often no pain, at least none of that severe pain which ushers in all varieties of the disease hitherto described. Not unfrequently the comparatively slight shortness of breath under which the patient labors escapes the notice of the patient himself, and he only seeks assistance of a physician because he "for some time past has become aware of a falling off in strength, and of having become pale and thin;" or he perhaps may think that he has some chronic disease of the abdomen, the more so as, in pleurisy of the right side, the depressed position of the liver may cause the right hypochondrium to bulge, and create tension in that region. Every physician in good practice must have seen cases of this kind, in which the patient has never been confined to the house, where he is unable precisely to fix the date of the commencement of his attack, and in which physical examination demonstrates the existence of enormous quantities of effusion in the pleural cavity. The extreme prostration and debility of these patients are easy of explanation, when we consider tha tthey are seldom free from fever, and that their pleuræ are filled up by an exceedingly albuminous effusion, which may amount to a weight of twelve or fifteen pounds. Such an effusion, under the most favorable circumstances, would only be very slowly reabsorbed; but it is very apt, as before said, alternately to decrease and to be reproduced, and finally, as we shall see, it terminates in most cases in consumption of the lung.

*Pleuritis with purulent exudation, empyema, pyothorax*, when it occurs by the gradual multiplication of young cells (which are never entirely absent in any case, in effusions of the form already described), can hardly be diagnosticated otherwise than by the long duration of the disease. The symptoms of compression, etc., are just the same as in effusions containing little pus. As already mentioned, pleuritic effusions often form during septicæmia and other diseases arising from blood-poisoning, in which an abundant cell-formation takes place from the commencement. However, it is not on account of its insidious attack, but owing to the serious implication of the system and to the blunted condition of the sensorium, that patients frequently make no complaint whatever, so that all subjective symptoms are wanting, and we must rely upon the objective ones.

With regard to the termination of pleurisy, all forms of the disease may end in recovery. Adhesions of the pleural surfaces, which always or nearly always remain, are hardly to be regarded as rendering the recovery incomplete, as patients may attain a very great age without suffering any serious inconvenience on this account. It has already been mentioned that the reabsorption of large effusions, even if rapid at first, is apt to be extremely tedious toward the last. We must be careful of diagnosticating a diminution of the exudation in all cases where the line of dulness sinks in the chest. A decrease of the dulness may also be due to the fact that the thoracic wall and intercostal muscles have become more yielding, or that the diaphragm has become relaxed and forced farther downward. These facts must always be borne in mind in judging of the condition of the patient. An obstinate exudation, which is very hard of reabsorption, should not be despaired of too soon, as its absorption may at last take place after we have given up all hopes of such an event.

When the compressed lung, either being enclosed in a firm fibrinous sheath, or its alveoli being occluded or adherent, is no longer able to admit air and to expand, and when the thorax collapses, the neighboring organs being employed to fill up the vacancy arising from absorption of the effused liquid, the pleurisy must be regarded as terminating in incomplete recovery. In persons thus affected, if otherwise in good health, the remaining portions of the lung can always oxygenate the blood sufficiently, and eliminate the carbonic acid, as long as the patient abstains from an overactive bodily exertion; and, notwithstanding that a part of the pulmonary capillaries has perished, the right side of the heart, which is then always somewhat hypertrophied and dilated, is still capable of so accelerating the current of the blood in the sound parts of the lung as to avert derangement of the circulation.

When an empyema "points" or opens externally, an œdematous swelling of the integument makes its appearance, not, however, at the most dependent part of the chest, but generally in the neighborhood of the fourth or fifth rib. Soon a hard, firm tumor protrudes through the intercostal space, which, after a time, begins to show fluctuation, and finally discharges a large amount of pus. This termination very rarely results in complete recovery, and in reinflation of the lung and reoccupation of the space restored by discharge of the pus. It is much more common in such cases for the thorax to collapse, and for secondary displacements of the organs to occur. Still more commonly there remains an imperfect closure of the thoracic opening (after pointing of an empyema), and a thoracic fistula forms, from which pus constantly flows, either in a continuous stream or in occasional profuse gushes. A patient with such a fistula may live for many years.

When empyema points inwardly, that is to say, into the lung, the perforation is sometimes preceded by the symptoms of a slight pneumonia, bloody sputa, a renewal of the stitch in the side, etc. At other times it takes place without warning, the patient suddenly discharging an enormous amount of purulent sputa after a violent fit of coughing. Here, too, in very rare instances, recovery with or without retraction of the thorax may ensue, but symptoms of suffocation, or of pyopneumothorax, are the more usual result (see Chapter III.).

Perforation of empyema through the diaphragm, or into neighboring organs, produces violent peritonitis, and the signs of abnormal communications, upon the details of which we could not enlarge without undue prolixity.

A fatal result in recent pleurisy most frequently arises from collateral hyperæmia, leading to intense œdema in the otherwise healthy portions of the lung. Rattling sounds, frothy and often bloody sputa, and great dyspnœa arise; carbonic-acid poisoning soon commences to develop, the sensorium of the patient becomes benumbed, and, with the general collapse, the action of the heart is weakened, the pulse grows small, the extremities cool, and the sufferer soon expires.

In other cases, compression of the lung and its capillaries gives rise to incomplete filling of the left ventricle, and to engorgement and obstruction of the right ventricle and the veins of the aortic system. This imperfect filling of the aortic system frequently
only to a small pulse, but to an exces-

of the urine (*Traube*). Distention of the veins leads to cyanosis and dropsy. Finally, owing to obstruction to the outflow from the renal veins, albumen, blood, and fibrinous cylinders frequently appear in the urine.

In other cases death ensues in consequence of bursting of empyema into the lungs, abdomen, etc. Death results still more frequently in unabsorbed effusions, in consequence of persistent, although moderate, fever, which consumes the organism, and which, therefore, is called hectic.

Finally, and, indeed, most commonly of all, tedious or imperfect absorption of empyema results in tuberculosis, or in chronic destructive pneumonia, the patient succumbing to the symptoms of consumption.

PHYSICAL SIGNS OF PLEURISY.—*When the exudation is scanty*, forming a thin membranous coating upon the pleural surfaces, or when it is liquid, and sinks to the more dependent part of the pleural sac, without, however, materially encroaching upon its space, the results of *inspection* usually are negative. It is only when respiration is extremely painful that we can perceive that the patients spare the affected side, and that its respiratory motion is not as free as upon the other side.

When *the pleuritic effusion is large*, inspection reveals a series of appearances, depending upon the fact that the inner surface of the chest is no longer affected by the traction of the elastic lung (as it should be), but is exposed to the pressure of the exudation.

1. The intercostal spaces over the area of the effusion are no longer shallow grooves, but are upon a level with the ribs, "they are effaced," and, indeed, are sometimes somewhat prominent.

2. Where the effusion fills up the entire pleura, the affected half of the chest appears enlarged in all directions, but chiefly in the line of the vertebro-mammillary diameter. When the effusion is not so large, when it is usually incapsulated in the posterior and lower regions of the pleural sac, dilatation of the thorax is limited to the region which contains the effusion. Very much more rarely, incapsulated exudations in the pleura produce prominence of some other portion of the thoracic wall.

3. In effusion of the left side, displacements of the heart can often be made out by inspection alone, and it is the same with displacement of the liver when the effusion is upon the right side; in the former case the impulse of the heart being too low, and too much toward the median line (sometimes, indeed, being perceptible to the right of the
m); in the latter, the right hypochondriac region shows an un-
rominence. Besides this evidence of pressure from within,
effusion upon the surrounding parts, inspection shows

that, as far as the effusion reaches, the thoracic wall does not take part in the respiratory movement. This is, in some degree, owing to infiltration and palsy of the intercostal muscles from collateral fluxion, and partly because the dilatation of the chest is physically impossible when the lung cannot expand. If the diaphragm be so much depressed as to form a projection into the abdomen, and if its muscles be not paralyzed, the contraction of the organ, with every inspiratory act, tends to flatten the convexity, which now, of course, is on its lower surface, so that, in these very rare instances, the epigastrium, instead of rising, sinks, during inspiration, upon the side where the effusion is situated.

If, as absorption of a pleuritic effusion progresses, the compressed lung again undergoes perfect expansion, there generally remains no sign of the disease which has just passed away. When the absorption is complete, the intercostal spaces again form shallow furrows, being once more exposed to the elastic traction of the lung. The dilatation of the thorax is corrected, the derangement of the respiratory movements has ceased, and the dislocated heart and liver have returned to their proper situations. Sometimes, however, after perfect absorption of the effusion, the heart, having become fixed by adhesions, remains out of place. If, however, the lung do not expand as the effusion becomes absorbed, all the dimensions of the chest seem to undergo reduction, and, more especially, its length and antero-posterior diameter, the ribs coming close together, and even overlying one another. The more the thorax loses its rounded form, and the more it becomes flattened, so much the more is its capacity diminished, even although its circumference remain the same. Hence, in cases where absorption of the exudation has commenced, if we wish to watch the progress of the reabsorption, and of the restoration of the lung, it is urgently recommended not only, from time to time, to measure the two halves of the chest, but to ascertain the length of the two vertebro-mammillary diameters by means of the callipers, and to compare the results of the two measurements. A still surer method is to draw accurate ideal sections of the two halves of the thorax, by means of the *Kyrtometer* of *Woillez*, which can be laid one upon the other and accurately compared at leisure. The more the ribs of the affected side are pressed together, so much the lower will the shoulder of that side descend, and so much the greater is the curvature of the spine. The collapse of one half of the chest, the depression of the shoulder, and the lateral curvature of the spinal column, the convexity of which is toward the sound side, are often so great as seriously to deform the patient, who is said to be "grown out of shape."

Finally, in cases where the lung has not reëxpanded after absorption of the effusion, inspection will often show that the heart beats far to the left, and even as far as the axillary line. The cause of this is that the heart, which at first was pushed to the right by the pleuritic effusion, upon reabsorption of the latter, is now drawn as far into the left pleural cavity, in order to fill up the vacant space caused by the disappearance of the liquid.

We may finally observe that the restoration of the normal dimensions of the chest, and even the secondary contraction of a chest previously distended by effusion, is not, by itself, a sufficient proof of the complete absorption of the exudation. A compressed lung occupies very little space, and, even after the pleural cavity has been greatly reduced in size, there is always room for a considerable quantity of liquid effusion. Upon *palpation*, a sensation of friction is perceptible in a large number of cases of pleurisy. The characteristics and peculiarities which distinguish this sensation from other sensible signs, as well as the conditions under which it arises, will be discussed while treating of the auscultatory phenomena.

Palpation, moreover, often furnishes important diagnostic signs of pleurisy with profuse effusion, from the peculiar character which the vocal fremitus exhibits in cases of pleuritic exudation. In general terms, it may be asserted that the pectoral fremitus is much weakened, or entirely suspended, wherever a liquid pleuritic effusion is in contact with the thoracic wall; but, above the limit of the effusion, where the compressed lung touches the side of the chest, the fremitus is intensified. It is quite manifest that a profuse liquid effusion will impede the conduction of sound-waves to the thoracic wall, and that it will also act as a powerful damper upon the vibrations of the latter, and it is equally plain that the retracted pulmonary tissue forms a better conductor for the passage of the vibrations to the chest-wall, and disturbs them less, than does the normal unretracted lung. As, under normal conditions, the vocal resonance is more plainly felt upon the right side of the chest than upon the left, feebleness or absence of pectoral fremitus upon the right side is of greater diagnostic importance than the occurrence of the same symptom upon the left side. In the anterior and lateral regions of the chest, the abrupt transition from absence to exaggeration of the fremitus is a valuable means of determining the limit of the exudation. Posteriorly, however, the signs change in a more gradual manner. According to some very accurate observations of *Seitz*, when the exudation is slight, the fremitus is only more or less weakened; when it is extensive, the fremitus is lost over the lower portion, but over the upper it is merely lessened, and even this diminution decreases gradually toward the level of the liquid. When the patient has a weak, high-pitched voice, whose wave-sounds hardly reach the thoracic wall under any circumstances, we lack an important aid to the diagnosis of pleuritic effusion.

Finally, palpation is of use in ascertaining the existence of the displacements of the heart and liver, alluded to in speaking of inspection, and which result from the effusion and its subsequent reabsorption. In cases of copious exudation into the right pleura, the edge of the liver may often be felt several fingers' breadth below the border of the ribs, or even lower.

*Percussion* affords no information of the presence of exudation when it is scanty and lies upon the pleura in the form of a thin, coagulated coating. On the other hand, large effusions, by which a considerable part of the lung is separated from the diaphragm and thoracic wall, furnish very characteristic signs upon percussion: 1. Over the region where the bulk of the liquid effusion lies in contact with the side of the chest, all vibration is checked, and the percussion-sound is dull. 2. Over the space into which the retracted lung (which, however, may still contain air) touches the thoracic wall, percussion is hollow and tympanitic. The conditions under which the dull, hollow, and tympanitic sounds arise have already been fully and repeatedly explained. No disease is better adapted for the demonstration of the difference between the dull and the hollow percussion-sounds than pleurisy with copious effusion. The dulness proceeding from pleuritic effusion generally first becomes perceptible in the region of the back and below the scapulæ. As it ascends it spreads toward the front. The dulness scarcely ever extends as far upward in front as it does behind. In many cases the dulness which reaches far up the back is not found at all over the breast, but only reaches as far as the axillary line. At other times, when nearly the whole pleural sac is occupied by the effusion, the upper boundary of the dull sound is but little lower in front than behind. Anteriorly, the dull percussion-sound changes abruptly to the empty tympanitic sound; posteriorly, as the upper limit of the effusion is approached, the dulness gradually becomes fainter and less distinct. The reason for this is, that the thickness of the body of effusion upon which the dull sound depends gradually diminishes from below upward. The form and boundaries of the dulness are not generally altered by changing the attitude of the patient, as agglutination and adhesions soon form about the effusion, which, although they still allow the pleural surfaces to slide upon each other, oppose their separation by the pressure of the exudation.

Upon *auscultation*, *friction-sounds* are heard whenever the surfaces of the pleura lose their smoothness through fibrinous deposit or the growth of rugged vegetations; but of course these sounds are only audible when the roughened surfaces are in contact, and when the respiratory movement causes them to rub together with a certain degree of rapidity. They are usually perceptible both upon inspiration and expiration, and give a distinct impression of scraping or of scratching, calling to mind the creaking of new leather, and there are often little jarring interruptions. It is most liable to be mistaken for a buzzing rhonchus, which is likewise often perceptible to the touch. A friction-sound, however, is scarcely ever as loud as a rhonchus, and, besides, is not altered by coughing; whereas a rhonchus almost always ceases after a vigorous cough, or, at all events, undergoes a change. It is also somewhat characteristic of a friction-sound, that it is heard more distinctly when the stethoscope is pressed rather firmly against the chest. This sound is rarely heard in the beginning of the disease, as the fibrinous deposit is not rough enough at first, and the patients, while they continue to suffer pain, breathe cautiously, so that the pleural surfaces do not rub together with sufficient quickness. The time at which it is audible most frequently is when the exudation begins to be reabsorbed, when the faces of the pleura, which previously were separated by the serum, now once more come into contact. They also become audible after evacuation of the liquid by tapping.

When the exudation is not very large, faint vesicular breathing, transmitted by the surrounding parts, can be heard over the whole region of dulness. When the effusion is very profuse, and when not only the air-cells but the bronchi are compressed by it, no respiratory murmur whatever is heard over the dull region, or, at the utmost, the sound is very faint and indistinct. It is only between the scapulæ and the spinal column, where the compressed lung lies close to the thoracic wall, that we can hear a feeble bronchial respiration and a faint bronchophony, the latter sometimes having a bleating tone, known as *ægophony*. In a few instances, where there is severe dyspnœa, in spite of the compression of the lung, and although we are obliged to suppose that the greater part of the bronchi are compressed and do not contain air, loud bronchial breathing is heard over the whole chest, even at points where there is a large mass of liquid between the ear and the lung, that is to say, at the sides of the thorax. Over the uncompressed lung, both upon the diseased and healthy sides, the respiration is loud and puerile, unless it be the seat of collateral hyperæmia and catarrh, when rhonchi and *râles* are to be heard.

Of course, the physical signs of pleuritis are greatly modified whenever old adhesions of the pleura prevent the exudation from collecting in the most dependent part of the chest. It would lead us too far to detail all these modifications, and we shall merely state that incapsulated effusion of very considerable magnitude may form between the diaphragm and the base of the lungs, and be very difficult of recognition, and often remain quite unrecognizable.

Diagnosis.—It is not always easy to distinguish a pleuritis with abundant exudation from a pneumonia, and the following are the chief points upon which we may rely for the purpose: 1. Pleurisy scarcely ever begins with a single violent chill, while in pneumonia this is the rule. 2. The course of a pleurisy is never so cyclic, nor is there that sudden and complete change for the better, or crisis, which we observe in pneumonia. 3. In pleurisy the sputa are indicative of catarrh or of œdema, and sometimes contain streaks of blood; but there never is that peculiar tough expectoration, stained yellow or yellowish-red, by intimate admixture of blood, which is pathognomonic of pneumonia. 4. The principal physical signs indicative of pleuritic exudation are, dilatation of the thorax, effacement of the intercostal furrows, displacement of the heart and liver, faintness or absence of pectoral fremitus, absolute dulness upon percussion, feebleness or absence of the respiratory murmur; whereas, in pneumonic infiltration, the chest is not enlarged, the intercostal spaces are not effaced, the heart and liver retain their situation, the pectoral fremitus is seldom enfeebled, and, indeed, is often intensified, the dulness upon percussion is not so absolute, and the respiratory murmur is almost always bronchial.

Patients having pleuritic effusions in their right side are not unfrequently supposed to have disease of the liver, and when we have ascertained by palpation that the liver reaches below the border of the ribs, and fills up the right hypochondrium, it is important that we should be able to tell whether the organ is enlarged or merely depressed. The following are the points of distinction between the two conditions: 1. The liver rarely pushes the diaphragm upward; hence, when the liver extends below the border of the ribs, and we at the same time find a dulness in the thorax which reaches farther upward than the normal hepatic dulness should do, we may reasonably infer that there is an effusion in the pleura and that the liver is pressed downward. 2. In the very rare instances in which, through enlargement of the liver (usually from an abscess, or a cyst of echinococcus), the diaphragm is abnormally pressed upward, and made to project into the cavity of the thorax, the dulness reaches farther up in the front of the chest, while in nearly every case of pleuritic effusion the opposite condition obtains. 3. When the liver is enlarged, its lower border, and with it the line of percussive dulness, moves downward upon inspiration and upward upon expiration. This does not take place when there is large effusion in the pleural sac, as the diaphragm is then depressed, and kept in a state of permanent expiratory extension. 4. The transition from the feeling of resistance, presented by the thoracic wall, to that produced by the enlarged liver, is immediate; whereas a small yielding interval is usually discoverable between the border of the ribs and the surface of a liver which has been displaced downward. 5. In enlargement of the liver, the lower ribs not unfrequently are somewhat bowed outward, the intercostal spaces, however, still remaining uneffaced, excepting in the rare instances in which a huge cyst of echinococci, or an abscess of the liver projecting far into the thoracic cavity, lies in contact with the side of the chest.

The main point of distinction between a small pleuritic effusion of the left side and an enlarged spleen consists in the change which takes place in the line of dulness during respiration, and which does not occur in pleurisy, but is easily perceptible in enlargement of the spleen.

Finally, the persistence of the fever, the emaciation and pallor of the patient, may awaken the suspicion that phthisis is developing. It should not be forgotten that both fever and wasting may be solely dependent upon a latent pleurisy, but the threatening phantom of incipient consumption should always be kept in view, and physical exploration of the thorax should be repeated again and again.

Prognosis.—Dry pleurisy is an altogether insignificant affection, nor does pleurisy with scanty sero-fibrinous effusion, of itself, ever cause danger, although the pain which attends it, being a main cause of the dyspnœa, augments the danger from the pneumonia or tuberculosis, or whatever the primary disease may be. Among the varieties of pleurisy with profuse sero-fibrinous effusion, that which runs an acute course from the outset admits of the most favorable prognosis. When the malady is of a creeping, insidious type, the prospect is much more grave, as, even after complete absorption, tuberculosis frequently appears as a sequel. This is also true of empyema when it develops from the foregoing variety, while the effusions which are purulent from the commencement involve a bad prognosis, from the nature of the diseases which give rise to them, namely, septicæmia, puerperal fever, etc.

Decrease of the effusion is to be regarded as a favorable sign, in the diagnosis of which, however, we must beware of the sources of error already alluded to. As there is more or less danger from consumption in the majority of cases, it is to be regarded as of favorable augury when the patient possesses a vigorous constitution. Finally, the earlier reabsorption commences, so much the more reason have we to hope that the lung may expand again, so that no deformity of the thorax may remain behind.

Symptoms of œdema of the lung and imperfect decarbonization of the blood at the commencement of the disease are to be viewed as unfavorable prognostic signs; as is also a diminution in the amount of urine secreted, which indicates that the arteries are incompletely filled. Still worse are the symptoms of over-distention of the veins, with cyanosis, dropsy, and the appearance of albumen, casts, and blood in the urine. The longer the effusion lasts, the more persistent the fever which accompanies it, the greater the wasting of the patient, so much the worse should our predictions be. Finally, all sequelæ, other than that of reabsorption, must be considered as prognostically unfavorable, although, as shown above, the danger may vary in degree.

TREATMENT.—The causal indications can no more be met in treatment of pleurisy than they can in treating pneumonia. In fact, even if we were aware that an attack of pleurisy were caused by "catching cold," if the fever were at all intense, a treatment by diaphoresis would be absolutely injurious.

*Indicatio morbi.*—The "antiphlogistic system," with its general and local blood-letting, its exhibition of calomel, and inunction of mercurial ointment until salivation is produced, and its subsequent derivation by blistering, etc., which formerly used to be the general practice in the treatment of pleurisy, but which, in the last ten years, has gradually fallen into discredit, recently has again been urgently recommended by *Joseph Meyer*, in a work which gives evidence of great industry. The arguments of this author in favor of the former method of treatment, and against the less active modes of procedure, are, however, based upon a very slender foundation. Thus great weight is laid upon the fact that a certain number of patients, with large pleuritic exudations, have been received into the Berlin Charité Hospital, who have not been bled, and who have never taken any mercury; and it is inferred that the profuseness of the effusion is a consequence of a neglect of active treatment. The enumeration of such cases as these proves nothing, unless the number of cases who were not bled, and yet who did not have large effusions, and who therefore did not seek admission at the Charité, be also given. But even the somewhat limited number of cases observed by *Myer* and others, in which recent cases of pleurisy, being treated by copious blood-letting, did not result in effusion, have not converted me. The assertion that pleurisy which is ushered in and accompanied by very acute symptoms, if left to itself, almost always terminates in profuse effusion difficult of reabsorption, I consider as quite erroneous. Indeed, the greatest danger in this respect is to be apprehended from the pleurisies which come on in a manner almost imperceptible, and whose duration is extremely tedious.

I still believe that venesection can be dispensed with in the treatment of pleurisy, with exception of a few rare cases, where certain symptoms demand it. I am convinced that it neither cuts short the malady, nor prevents the effusion; and, as this disease, owing to its tedious course, is always liable to lead to deterioration of the blood, and to consumption, I regard the practice of bleeding as still more dangerous in pleurisy than in pneumonia.

At the commencement of an attack of pleurisy, however, I cannot sufficiently recommend the use of cold and of local blood-letting. It is highly essential that the proper moment for employing this important treatment should not be neglected, as much evil may then be prevented, which, at an after-period, is difficult to repair. When the patients dread the application of cold compresses, or if the latter do not relieve the pain and dyspnœa in an hour or two, a tolerably large number of leeches, or cut-cups, should be applied; and, if the pain, which is almost always relieved by the depletion, recurs in the course of a day or two, we should not hesitate to repeat the local blood-letting until the relief becomes permanent.

Besides this, and for want of remedies of more certain action, half a drachm of mercurial ointment may be rubbed into the affected side of the chest twice daily, but the inunction must be at once suspended the moment that signs of mercurial sore mouth appear. As a decided benefit is sometimes obtained from the inunction of mercurial ointment in inflammation of other serous membranes, particularly inflammation of the articular capsules, its efficacy should always be tested in cases of recent pleurisy, although its action is then far less easy to observe.

Having convinced myself, from my own observation and from the cases reported in the work of *Meyer* above alluded to, that the fever is not materially aggravated by the use of blisters, I now retract my former advice not to resort to vesication while fever lasts. Indeed, the application of large vesicatories seems to be of service in certain cases, but, when used at all, they must be used early.

In protracted cases, large hot poultices, which, however, must not be too heavy, do good service.

Internal medication, save when called for by special symptoms, is unnecessary in treatment of pleurisy. The antiphlogistic action of nitrate of potash, of tartar emetic, and of calomel, I regard as highly problematic; and, moreover, the exhibition of calomel is not without its dangers, as it tends to augment the impoverishment of the blood, and the tendency to exhaustion which already exists.

*Indicatio Symptomatica.*—Antipyretic treatment is always proper when the fever is very high at the commencement of the attack, or when it is so persistent that there is reason to fear that it will exhaust the patient. With this object in view, the very customary use of digitalis is to be recommended in certain cases. It does not affect the primary disease at all. In recent cases, where the fever is high, I usually give digitalis in the form of infusion (℈ss to ℥vj); in tedious cases, where the fever is of a more latent character, I give it in substance (gr. j for a dose, usually combined with equal parts of quinine).

Dyspnœa, when it arises from collateral hyperæmia of the uncompressed parts of the lung, especially when signs of commencing collateral œdema already exist, imperatively demands venesection. Under such circumstances I have often ordered three or four successive bleedings, and as I did not bleed to cure the pleurisy, but on account of the danger arising from the hyperæmia, I have not laid myself open to the charge of inconsistency.

It is far more rare for cyanosis, dropsy, and other symptoms of venous engorgement of the aortic system depending upon disturbance of the pulmonary circulation, to require venesection.

Especial attention is to be paid to the deterioration of the blood, which often becomes apparent at an early period, owing to the volume of the effusion, and to the wasting induced by the fever. There should be no hesitation about administration of the ferruginous preparations, and of a nourishing diet. The old prejudice, that iron causes congestion, is entirely void of foundation.

Remedies for the promoting of reabsorption of the effusion deserve little reliance. It is, indeed, questionable whether it be possible, by any therapeutic means, to bring about the conditions upon which the absorption of pleuritic effusion depends. If, after the inflammatory symptoms have subsided, the effusion remain undiminished, all medication, both external and internal, is to be rejected, and the application of blisters is of very doubtful use. The fact that pleuritic or other pathological effusion has been rapidly absorbed during an attack of cholera, when the blood had become thickened by loss of its water, makes it seem rational to attempt to reduce the water of the blood by the administration of diuretics and drastics, in order to promote absorption of the effusion. Unfortunately, the action of the diuretics, of which bitartrate of potash, boracic cream of tartar, and the juniper-berry are the best, is very uncertain, so that we cannot promise ourselves much from their use, and the pernicious effect of the drastics upon digestion and assimilation forms a serious objection to their employment. In one case, which I did not treat myself, but which I watched with attention, a pleuritic effusion, which had withstood all efforts to remove it, diminished rapidly under what is known as *Schroth's* treatment; the physician in charge, conceiving the idea of producing inspissation of the blood by diminishing the supply of water coming to it, instead of attempting to abstract the water from it, fed the patient upon the driest possible diet, and almost entirely deprived him of drink. I have seen other cases, where this treatment failed, as, contrary to all reason, the patient was allowed a copious supply of wine on certain days. An attempt may also be made to excite reabsorption by the outward and inward administration of iodine, which has a well-known reputation as an absorbent. I have seen such remarkably rapid absorption take place under the internal use of syrup ferri iodidi (ʒ ij) with syrup simplic. (℥ ij), a teaspoonful being taken every two hours, in conjunction with the external application of a weak compound solution of iodine (iodini. ʒ ss, potass. iodid. ʒ ij, aqua destil. ℥ ij), upon the affected side of the chest, that I cannot help regarding the beneficial action of this prescription as probable, although I do not regard it as proved.

Considering our slender ability to excite or even to hasten reabsorption of pleuritic effusions by means of internal medication, the discovery that their evacuation by surgical means is attended by much less danger than was formerly supposed, and the frequent and early practice of such operations in cases of pleurisy with effusion, must be considered an important advance in therapeutics. It must be evident that every additional day, during which the lung is exposed to pressure, and the longer the time allowed for cells to multiply in the exudation, so much the more are the chances of complete recovery diminished, and the danger of a fatal termination increased. Regarding the precise indications for the procedure of tapping and for its various modifications, as well as regards the details of the operation, we refer to the hand-books of surgery.

## CHAPTER II.

### HYDROTHORAX.

Etiology.—Hydrothorax is not the result of an exudation, but of a dropsical transudation into the pleural sac. In most instances its source is easily traceable to one or other of the well-known conditions under which pathological transudations arise, namely, increase of the lateral pressure within the veins, and decrease in the amount of albumen in the serum of the blood, the so-called dropsical crasis.

"Water on the chest," which, to the minds of the laity, is one of the most formidable of maladies, in which view the older pathologists participated, is never an independent and primary affection, but is always secondary, being the result of some morbid process, which has given rise to the conditions necessary for the production of a pathological transudation. Hydrothorax, therefore, no more deserves the name of a disease than does dropsy of the subcutaneous tissue, or dropsical effusions into other large cavities of the body. It is merely as a matter of convenience, and in compliance with ancient custom, that I have included it in the list of disorders of the pleura.

Hydrothorax, caused by augmented pressure within the veins of the pleura, is one of the more formidable accidents occurring in diseases of the lung which (as we have shown in a previous chapter) obstruct the action of the right side of the heart, and produce venous engorgement of the aortic circulation. It occurs with equal frequence in certain diseases of the heart, namely, derangement of its valves and degeneration of its muscular substance, which, as we shall show by-and-by, also impede the outflow of the blood from the right side of the heart, and from the veins of the aortic system.

Hydrothorax, dependent upon diminution of the amount of albumen in the serum of the blood, whose pathogeny is somewhat obscure, as we shall show while treating of Bright's disease, is one of the accompaniments of grave cachectic conditions, and especially of inflammations occurring in chronic degeneration of the kidney with albuminuria, in malignant malarious disease, and in dysentery of long standing.

Whether hydrothorax be a consequence of venous engorgement, or of a morbid state of the blood, it is usually but one of the symptoms of a general dropsy. When arising from the former condition, it sometimes precedes the effusion into other cavities, whereas it almost always appears at a late period when due to the latter.

Anatomical Appearances.—Hydrothorax is almost always double, but one pleura sometimes contains more liquid than the other. Its amount varies from a few ounces to many pounds. It usually is movable, but is sometimes incapsulated by old adhesions. The transudation contained in the pleura is a clear, yellowish liquid, consisting of water, albumen, and the salts of the serum of the blood. It is easily distinguishable from a pleuritic effusion, by the absence of the fibrinous coagula and inflammatory changes in the pleural surfaces. The latter have lost their polished appearance, and have a milky opacity, and both they and the subserous tissue are slightly swollen by serous infiltration. When the effusion is very large, the lungs, unless they are held down by old adhesions, are driven up against the spinal column, large portions of them being in a state of compression.

Symptoms and Course.—From the most ancient times down to the beginning of the present century, the symptoms and course of water on the chest, as an independent disease, have not only been described with great fulness and accuracy, but, in many instances, the diagnosis has been confirmed by *post-mortem* dissection. This was because the symptoms ascribed to the disease by the ancients apply to the class of affections of the lungs and heart which ultimately results in dropsical transudations of all kinds, and in effusions into the pleura, among others. There is no doubt that emphysema, a disease which formerly must have been as common as it is now, but the existence of which was entirely unknown and overlooked, *post mortem*, until the days of *Laennec*, was generally supposed to be a dropsy of the chest, and was described as such.

In the present state of science we know that such symptoms as severe dyspnœa, aggravated by every slight exertion, and which compels the patient to sit upright in bed, suddenly starting in terror from sleep, and œdematous swelling of the malleoli and eyelids, do not always denote the existence of hydrothorax, as similar symptoms occur from diseases of the lungs or heart, without dropsy of the pleura. But, as we know that hydrothorax is a very common event in diseases which give rise to these symptoms, and that the distress of the patient is greatly aggravated by such a complication, it behooves us to make repeated physical exploration of the chest, that we may be aware of the fact of its appearance.

Physical exploration of the chest likewise furnishes the only certain means of recognizing the existence of the hydrothorax occurring in Bright's disease, or in any other malady, accompanied by general dropsical cachexia, as the symptoms of dyspnœa, which attend its development and progress, are equally attributable to other sources, particularly to incipient œdema of the lungs. The physical signs of hydrothorax bear great similarity to those of pleuritic effusion, although the resemblance is not complete.

*Inspection* reveals a dilatation of the chest in the region of the transudation, but the intercostal furrows are not obliterated, since the intercostal muscles, not being paralyzed by collateral œdema, offer resistance to the pressure of the liquid. The liver, which is often enlarged by venous engorgement, is depressed when the effusion is large, but the heart is hardly ever displaced, as the pressure upon the mediastinum is usually tolerably equal upon each side.

*Palpation* gives an enfeeblement or total arrest of the pectoral fremitus wherever the effusion touches the thoracic wall, while above the effusion its intensity is increased.

The *percussion-sound* is dull over the effusion, above it it is hollow and tympanitic. The dulness does not extend itself in the peculiar manner which is almost pathognomonic of pleuritic exudation. When the patient stands or sits upright, its upper limits are upon the same level both before and behind. Moreover, the boundaries and shape of the area of dulness change slowly when the patient alters his attitude. Upon auscultation over the region of dulness, the respiratory sound is weak, indistinct, or even absent. Between the scapulæ and the spine there is feeble bronchial respiration.

TREATMENT.—Treatment of hydrothorax is identical with a treatment of the main disease, and, as usually but little can be effected to benefit the latter, our success is seldom good. When the dyspnœa is very intense, and is manifestly dependent upon the profusion of the transudation, its evacuation by tapping is indicated. The benefit temporarily obtained in such circumstances is often very marked.

## CHAPTER III.

### PNEUMOTHORAX.

ETIOLOGY.—We are not at liberty to suppose that under any circumstances gases are secreted by the surfaces of the pleura and collect in its sac. It is manifest that the facts, upon which such a theory of the origin of pneumothorax is based, have been falsely interpreted. I will not absolutely deny that decomposition of a pleuritic effusion may give rise to the development of gases in the pleura without the entrance to it of air; but such occurrences are, at least, very rare. The most common source of pneumothorax is the entrance of air into the pleural cavity through an opening in the pulmonary surface, or through perforation of the thoracic wall.

Perforation of the pulmonary pleura may take place from within, the destructive disease of the lung attacking the pleura; or from without, an injury or gradual degeneration of the tissues upon the surface of the pleura penetrating the lung. It is by the first of these methods that pneumothorax arises in pulmonary abscess, gangrene of the lung, and consumption. Most of the cases of pneumothorax, which have been reported, have occurred in phthisis (not in tuberculosis, as is commonly said), in consequence of the opening of a vomica into the pleural sac. I would here remark that a tedious chronic consumption is far less liable to result in pneumothorax than a consumption whose progress is rapid and subacute. When the disorganization of the lung proceeds slowly, the pleural surfaces usually become firmly adherent to one another as the morbid process approaches the surface, so that, if perforated, air cannot enter the cavity of the pleura. Quite often the pneumonia, upon which the consumption depends, is of recent origin; no dulness, upon percussion, or bronchial breathing, at the apex of the lungs, as yet being discoverable, the strength and nutritive condition of the patient having suffered but slightly when the pneumothorax forms. The rapid destruction of but a single lobule in a state of caseous infiltration, and lying at the surface of the lung, might induce the catastrophe. Besides the pneumothorax, arising from destructive disease of the lung, there are other rare but well-authenticated cases in which the disease has resulted from a rupture of dilated emphysematous subpleural pulmonary vesicles.

The majority of cases of traumatic pneumothorax are not, as a rule, the result of entrance of air into the pleural sac, through a mere penetrating wound of the thoracic wall, but of a stab or gun-shot wound, which also involves the pulmonary pleura, thus permitting the escape of air from the lung into the pleural sac. In fracture of the ribs, it sometimes happens that the pulmonary pleura is lacerated by spicula of bone, causing pneumothorax, without perforation or appreciable wound of the wall of the chest. A gradual perforation of the pulmonary pleura, by ulceration of its external surface, is a much more frequent occurrence than this acute traumatic perforation. As we have already stated, the bursting of an empyema into the lungs and its evacuation through the bronchi take place in a similar manner. After a certain amount of pus has been discharged by the coughing-fit which follows the perforation of the empyema, a corresponding quantity of air naturally enters the sac with the next inspiratory act. The pyothorax is thus converted into a pneumo-pyothorax. In this form of disease the air scarcely ever passes freely into the pleura, but merely enters a space firmly bounded by adhesions, by which the empyema is incapsulated, and which separate it from the rest of the cavity, a condition to which we shall allude again while discussing the symptoms.

We have already stated that all perforations of the thoracic wall do not cause pneumothorax. If the track of the wound through the wall of the chest be tolerably narrow, and if its direction be oblique, the integument forms a sort of valve at its outer end, which prevents the air from entering the chest. It is the same with the fistulous passages, which usually remain after the spontaneous external opening of an empyema. On the contrary, if the thoracic wall be penetrated perpendicularly, and the opening be sufficiently large, pneumothorax does occur, air flowing in and out of the pleura, through the orifice, as the chest heaves and falls. It may be mentioned, finally, that in exceedingly rare instances, pneumothorax arises through the perforation of the pleural sac by ulceration or degeneration of growths within the stomach or œsophagus.

Anatomical Appearances.—The existence of pneumothorax may often be guessed upon simple inspection of the cadaver, owing to the enormous distention of one or other side of the chest, with obliteration or prominence of the intercostal spaces. If the abdomen be opened first, we find that the convexity of the diaphragm faces downward, and that the liver or the spleen is deeply displaced. If a knife or trocar be thrust into the distended side of the chest, the air gushes out with a hissing sound, capable of extinguishing a light held before the orifice. The gas which escapes consists principally of carbonic acid and nitrogen, and contains but little oxygen. Its quantity varies, but is usually sufficient to produce the monstrous dilatation of the chest above alluded to. It is rare for the pleural sac to contain air alone. Even though the patient only survive the pneumothorax a day or two, pleurisy develops, and we find sero-purulent or purulent exudation in the pleura besides the air. The quantity of the effusion is variable, and generally is largest when the pneumothorax is of longest standing. Again, the effusion may almost fill up the pleura, while the amount of air it contains may be very small. Finally, all the air may disappear from the sac, so that it contains nothing except the effusion.

Whether the pleura contain air alone, or both air and pleuritic exudation, the lung is compressed into a very small volume, completely void of air, and is pushed up against the spinal column. And it is only when it is partially attached by adhesions to the thoracic wall that it occupies any other position. In many cases it is only with great trouble, and by inflating the lung under water, that the point of perforation, which is usually covered by fibrinous deposit, can be discovered. In other instances the orifice is already closed. Besides the abovementioned downward displacement of the diaphragm, a considerable lateral displacement of the heart and mediastinum is generally found in pneumothorax.

Many important variations from the anatomical alterations described above take place when uniform retraction of the lung on all sides is prevented by extensive and firm adhesions of the pleural surfaces. Sometimes the air which has escaped is contained in spaces of small capacity, and enclosed by adhesions upon all sides. Then, only, the adjacent parts of the lung are compressed, and the thorax is only partially dilated; the liver and heart are not displaced. This latter condition is the rule in pneumothorax from perforation of an empyema, but it occasionally is met with also in cases of perforation of a superficial cavity.

SYMPTOMS AND COURSE.—The symptoms of pneumothorax are generally very striking and characteristic, and are easily understood, if we bear in mind the consequences which necessarily must ensue after perforation of the pulmonary pleura, or of the thoracic wall.

The pleural cavity being no longer hermetically sealed, the lung retracts by virtue of its elasticity. A retraction of the lung, such as we find upon opening the thorax upon the dissecting-table, occurs during life, at the moment when the air enters the pleural sac through a perforation, either externally or internally. Even the lung of the uninjured side retracts as far as the mediastinum can yield to the traction, now exerted upon one side of it alone. Immediately after the establishment of the pneumothorax, the cavity only contains the air which has been expelled from the lung by virtue of its elasticity. With the next inspiratory movement of the chest an additional quantity of air enters the pleural sac, and, if this air can pass out again with the succeeding expiration, the thorax returns to its expiratory state, and the lung does not suffer compression. If, however, the air which enters the pleura during inspiration cannot escape with the next act of expiration, the thorax remains dilated, and the lung is compressed. This process is repeated until the thorax attains the utmost dilatation which a forced inspiratory act can produce in it, and until all air has been driven out of the lung, and its compression is complete. Owing to the peculiar ragged character of the opening in the pulmonary pleura, through which the air enters the pleural sac in most cases of pneumothorax, it generally happens that the air which gets in cannot get out again. The orifice opens like a valve during inspiration, and is closed by the pressure of the compressed air when expiration commences. Finally, after the tension of the air within the pleura has reached a sufficient degree of intensity, the valve is permanently shut, even during inspiration, and, although there is no agglutination or adhesion of the point of perforation, no more air enters the cavity. In pure pneumothorax, the dilatation of the chest does not exceed the normal expansion which forced inspiration can produce in it. The extraordinary degree to which this normal limit is often exceeded, and the excessive distention which the walls of the chest frequently undergo, are due to a consecutive effusion of liquid, which also takes up room, or, in other words, the pneumothorax has become a pyo-pneumothorax. In the rare instances in which the air passes freely in and out of the thorax, as occurs in large wounds or fistulous openings which penetrate perpendicularly, and in spacious fistulous communications, with rigid resisting walls running between the pleural cavity and a large bronchus, there is no dilatation of the chest, and no compression of the lung, which, however, has generally been emptied of its air by other causes.

When a vomica perforates the pleural sac, the moment of perforation is usually distinctly perceptible to the patient. He feels as if "something had given way, or burst, in his chest." Immediately afterward a dyspnœa commences, which rapidly assumes the utmost intensity. The patient can only lie upon the affected side, or else is compelled to sit upright, so as to give the freest play possible to the sound side of his chest. This dyspnœa is partially owing to the sudden compression of one lung, and in part to the collateral hyperæmia and collateral œdema, and obstruction of the alveoli in the sound lung, consequent upon the compression of the vessels of the diseased one. In all the cases of sudden pneumothorax which I have seen, the patients soon began to complain of *severe pain* in the region of the lower ribs, which is to be attributed either to the strain upon the diaphragm, or else to the pleurisy which is excited by the escape of air, and of the contents of the vomica into the pleura. If the patient be not very anæmic already, marked signs of engorgement of the right side of the heart, which is deprived of half its efferent channels by compression of the vessels of the lung, soon are added to the above symptoms. The patient becomes cyanotic and dropsical; swelling of the face and extremities is often observable from the very commencement. The pulse is small, the urine scanty, the skin cool, partly because the left side of the heart receives blood from one lung only, and hence is not completely filled, partly in consequence of the collapse which accompanies bursting of a vomica into the pleural sac, and other serious injuries, such as the perforation of ulcers of the stomach.

Some patients die in a few hours, or even sooner, from the combined effects of want of breath and collapse. In other instances, death does not take place for days or even weeks. The collapse then subsides, the patient grows warm again; but the dyspnœa continues, and grows worse, as the pleuritic effusion increases, and pushes the heart and mediastinum more and more against the sound lung. The cyanosis and dropsy also increase. The patients finally succumb to pulmonary œdema and to insufficient decarbonization of the blood, or perhaps die of the consecutive pleurisy, exhausted by fever and by the profuseness of the effusion.

Recovery from pneumothorax is rare. When it occurs, the pneumothorax first changes into a simple pyothorax, the liquid exudation accumulating in the chest, and so augmenting the pressure upon the air contained in the pleural sac, that it is diffused among the adjacent vessels. Then, if circumstances favor, the effusion itself may be reabsorbed, and, if meantime the orifice of the perforation be closed, the lung may expand again. I treated a patient in Magdeburg, who, after lying for weeks in a condition of the utmost misery, so that her death was daily expected, so far recovered in the course of three months as to marry, and to carry on a somewhat extensive business. In other cases, a wide communication forms between the pleural cavity and some large bronchus, which still remains patulous in the compressed lung, and from time to time—but only when the patient assumes particular attitudes (as has been related in a very interesting case in *Romberg's* clinic, reported by *Henoch*)—a part of the liquid contents of the cavity which has entered the bronchi is discharged by coughing. When the perforation of a cavity causes the admission of air into a space which has been enclosed in old and firm adhesions, or where an empyema has pointed into the lung, and where air has entered to take the place of the pus which has been evacuated by the bronchi, the symptoms and course of pneumothorax are altogether different from those described above. In such cases, especially in the latter instance, there often are no subjective symptoms whatever, and the malady is only discovered accidentally when the chest of the patient is examined. When the quantity of air in the pleural sac is large, physical examination gives the following results:

*Inspection.*—If we find that a consumptive patient (whom a few days ago we left walking about, or whose attitude as he lay in bed was perfectly unconstrained) now exhibits signs of great dyspnœa, that he lies upon one side, anxiously avoiding all alteration of this position, these symptoms alone (particularly if the change have taken place suddenly) should awaken a strong suspicion that pneumothorax has been established. Upon looking at the chest, even an unpractised eye can mark the dilatation which it has undergone, the obliteration of the intercostal furrows, and the absence of respiratory movement upon the affected side, and, when the pneumothorax is in the left pleura, that the cardiac impulse is visible to the right of the sternum.

*Palpation.*—Displacement of the heart toward the sound side, as well as downward displacement of the liver, in pneumothorax of the right side, is still more perceptible upon palpation. The pectoral fremitus is generally quite imperceptible, and is always fainter upon the affected side than upon the sound one.

*Percussion.*—Over the region of the pneumothorax the percussion-sound is full, clear, and tympanitic, and when the pneumothorax is of the right side it extends too far downward, and when of the left too far inward. When the thoracic wall is very much disturbed, the intensity of the pressure hinders the formation of regular vibrations, and the sound is not tympanitic. So tense does the thoracic wall sometimes become, that percussion does not produce any sonorous waves at all, and, even when practised with heavy strokes, only gives rise to a feeble, dull sound. Great stress has recently been laid upon the change of pitch in the percussion-sound of pneumothorax occasioned by the patient's lying down or sitting up (*Biermer*), it being supposed that the diaphragm is depressed by the effusion, and the long diameter of the pleural sac is increased, while in the erect attitude (*Biermer*, *Gerhardt*). I cannot help doubting the constancy of this increase in the long diameter of the pleural cavity in the erect posture, and indeed believe that, sometimes, the very reverse occurs, that is, when there is a certain amount of exudation in the cavity. Finally, a metallic clang is often heard upon percussion, especially when the ear is laid upon the chest during the operation. The percussion-sound becomes dull, as far as the effusion extends, in a very few days. A characteristic sign of pyo-pneumothorax is, that the limits of the dulness change as the patient alters his posture. When he lies upon his back, the sound in front may be full, down to the border of the lower ribs; if he stands up, the dulness may extend far up his chest.

*Auscultation.*—When there are both air and liquid in the pleural sac, a distinct metallic splashing (like the sound of water, shaken up in a half-filled bottle) is often audible, even without putting the ear to the chest, whenever the patient suddenly changes his position, or when he is shaken (succussion). No vesicular respiration is to be heard, which, in conjunction with the full percussion-sound, is a symptom of great significance. In its stead, we hear metallic sounds and amphoric breathing, and especially the metallic rattling (the *tintement metallique*) sounds which are also heard over large vomicæ with smooth, regular, concave walls. We are not at liberty to infer, from the existence of metallic sounds, that air flows into and out of the pleural sac, as such sounds may also arise when the communication is closed, the murmurs generated in the lung giving a metallic reverberation. While the above signs arise with great completeness and harmony, in most cases where the air is capable of free movement in the cavity of the pleura, after perforation of a vomica, so that the affection is then very easy of recognition, many of them may be absent when the pneumothorax is incapsulated. If the space containing the air be too small, or too irregular in shape, to be capable of assuming a regular bulbous form under pressure of the air and exudation, no metallic sounds are heard, either upon percussion or auscultation. The most constant and trustworthy sign of incapsulated pneumothorax is a full percussion-sound, with absence of respiratory murmur.

Besides this, in a few cases, in which, after bursting of an empyema into the lungs, the cavity containing the air and effusion was extremely irregular, I have been able, by laying my hand upon the thorax, to feel the liquid wash against the anterior side of the chest, when the patient raised himself quickly and with energy.

DIAGNOSIS.—It is only when we are suddenly called to the bedside of a patient, or when we receive him into hospital in such a condition of suffocation that he is unable to give any account of his previous illness, that pneumothorax is liable to be confounded with emphysema. In all other instances, the rapid development of the dyspnœa in pneumothorax and its extremely gradual establishment in emphysema place the matter beyond a doubt.

In the less obscure cases we may rely upon the following tokens. 1. In emphysema, both sides of the chest, in pneumothorax but one is dilated (emphysema is bilateral, pneumothorax is almost always monolateral). 2. In emphysema the intercostal spaces are shallow furrows;

in pneumothorax they have disappeared, or become prominent. 3. In emphysema the vesicular breathing is weak, but is not absolutely wanting; or we hear the sounds of bronchitis in its place. In pneumothorax we never hear vesicular murmur, but metallic sounds are frequently audible. 4. In emphysema the pectoral fremitus is perceptible; in pneumothorax it is hardly ever present.

Pneumothorax is to be distinguished from large, empty, superficially situated cavities—over which upon percussion we hear the metallic ring, and in which amphoric breathing and metallic tinkling are audible upon auscultation—by the following points of difference: 1. The thorax is depressed over a large superficial cavern; over a pneumothorax it is dilated. 2. Where a cavity exists, the pectoral fremitus frequently is strengthened; in pneumothorax it is imperceptible. 3. The *râles* in a cavity are usually loud and numerous; in pneumothorax they are few and faintly audible. 4. Where there is only a cavity, the neighboring organs are not dislocated; in pneumothorax the displacement is generally well marked. 5. In a cavity the pitch of the tympanitic percussion-sound is altered by opening and shutting the mouth. This is not the case in pneumothorax.

TREATMENT.—The treatment of pneumothorax can only be a palliative one, and a treatment of symptoms. In many cases, particularly where the volume of the blood is not as yet diminished, as in the traumatic form of the malady, venesection may be urgently demanded at the outset. We may even be required to repeat the venesection, should the uncompressed lung become so intensely hyperæmic as to be unable to fulfil its function. The pain which arises from straining of the diaphragm and commencing pleuritis should be treated by local blood-letting and cold applications. Opiates are indispensable, for the mitigation of the distress of the patient, and to procure him rest.

Puncture of the thorax with a fine trocar is indispensable, as a means of relieving the dyspnœa of the patient, although the effect obtained is merely palliative. The operation does not benefit the lung of the affected side, but rather the sound lung, when the mediastinum is displaced by pressure of the accumulated air and exudation, and hence encroaches upon the unperforated pleura.

The principles laid down for the treatment of pleuritis are applicable to the treatment of the more advanced stages of pneumothorax.

## CHAPTER IV.

### TUBERCULOSIS OF THE PLEURA.

I. GRAY miliary tubercle of the pleura occurs almost exclusively in acute miliary tuberculosis, simultaneously with miliary tubercles of the lung, spleen, liver, and meninges. As we have already seen, the patient succumbs to the intensity of the fever in this malady, before the tubercles undergo further metamorphosis. Miliary tuberculosis of the pleura gives rise to no peculiar local symptoms.

II. Tubercular granulations develop with far greater frequence in the young false membranes which grow from the pleura after repeated relapses of pleurisy. We have seen that rupture of small blood-vessels is liable to occur during the inflammation which the new vegetations (profusely supplied as they are with large and delicately-walled capillaries) have to undergo in a relapse of pleurisy. This accounts for the hæmorrhagic character of the exudation, which accompanies tuberculosis of pseudomembranes. It appears in the form of numerous nodular prominences, of about the size of a hemp-seed, which at first are white, afterward acquiring a yellow color. It is this tuberculosis of pseudomembrane which is best adapted for the study of the origin and metamorphosis of tubercle (*Virchow*). The symptoms of this form of tuberculosis of the pleura are indistinguishable from those of a pleurisy with hæmorrhagic exudation.

## CHAPTER V.

### CANCER OF THE PLEURA.

Cancer of the pleura never occurs primarily, and only is met with in advanced general cancerous infection, and where cancer has arisen in other organs, and particularly in those adjacent to it. It most frequently complicates cancer of the mamma, of the mediastinum, and of the lungs, and arises with especial frequence after extirpation of cancer of the breast. The pleura is then either perforated from without, by cancerous deposits of the vicinity which sprout inward, in the form of bulbous swellings, or else independent cancerous nodules spring up upon the pleura, attaining the size of a fist, and presenting a lardy, marrowy appearance, and a nodulated or more or less level surface. Cancers of the pleura are full of cells, have very little connective tissue, and belong to the class of medullary sarcoma.

When the cancerous degeneration is somewhat extensive, a liquid collects in the cavity of the pleura, which, as it were, stands midway between inflammatory and dropsical exudation. It contains fibrin indeed, just as we find it in other serous sacs, which have become the seat of cancerous disease, but it does not coagulate until a late period; that is to say, we find no fibrinous deposit in the effusion, but, upon allowing the liquid to stand after evacuation, it gradually precipitates coagulating masses, often continuing so to do for days.—(Hydrops lymphaticus, *Virchow*)—(Hydrops fibrinous of *Vogel*.)

In the very great majority of cases, cancer of the pleura cannot be diagnosticated. Should an effusion gradually form in the pleural sac in a case of long-standing cancer of the breast, which is immovably attached to the thorax, or after extirpation of a mammary cancer, we are entitled to suppose that a cancerous growth exists upon the inner wall of the thorax. Large tumors may cause compression of the lung or greater bronchi; may displace the heart, or exert pressure upon the great vessels. Thus dyspnœa, cyanosis, and dizziness may arise, symptoms whose meaning, however, is seldom rightly interpreted. When large cancerous tumors reach the wall of the chest, the percussion-sound at the point involved becomes absolutely dull; and, if the tumor lie against the thorax posteriorly, with the aorta in front of it, pulsation may arise, and the cancer be mistaken for an aneurism of the aorta. This may happen all the more easily, as a spurious murmur may be produced in the aorta at the point where it is compressed, which may be perceptible at the feebly pulsating point of dulness. However, the pulsation as aforesaid is always weak and the false murmur is always merely a systolic one. We never hear the double, false murmur which we seldom fail to hear in an aneurism lying in contact with the chest. Finally, the history of the case, particularly that of previous extirpation of a cancerous tumor of the breast, will assist the diagnosis.

We cannot consistently speak of any treatment of cancer of the pleura, as we must confine our efforts to palliation of its more distressing symptoms.

# APPENDIX

TO THE

# DISEASES OF THE RESPIRATORY ORGANS.

## *DISEASE OF THE NASAL CAVITIES.*

## CHAPTER I.

### HYPERÆMIA AND CATARRH OF THE NASAL MUCOUS MEMBRANE—CORYZA, GRAVEDO, COLD IN THE HEAD.

ETIOLOGY.—The nutritive and functional disorders characteristic of catarrh are to be seen with extreme frequence in the nasal mucous membrane, where they are called "*cold in the head*" (Schnupfen). Far more rarely, and almost solely in the course of infectious maladies, the nasal mucous membrane is attacked by croupous or by diphtheritic inflammation.

The same remarks apply, with regard to the etiology of nasal catarrh, which we have already made in treating of catarrh of the larynx and bronchi. Predisposition to cold in the head varies greatly in different individuals. In general, it is greater in children and in feeble, delicate, and, above all, in scrofulous persons, than in adults and in individuals who are muscular and robust. Gradual "hardening" diminishes the predisposition, so that it is comparatively rare for inveterate snuff-takers, who are in the habit of irritating their nasal membranes almost constantly, to suffer much from catarrh. Besides these, however, there are sources of predisposition which are quite unknown to us, or, in other words, we find a remarkable tendency to coryza in persons in whom we are not able to detect any peculiarity by which they may be distinguished from other less predisposed individuals.

The causes which occasion catarrh of the nose are very numerous, and the almost universal impression which prevails among the laity, that "colds" always proceed from chilling of the skin, is erroneous;

although, indeed, most cases of coryza do depend upon exposure to cold, and particularly upon exposure of the feet.

Next in frequence, local irritants cause catarrh of the nasal passages, such as the entrance of hot air, dust, acrid gases or foreign bodies; or the snuffing of tobacco by persons unaccustomed to it; likewise blows or shocks which the nose may encounter, and often repeated and violent blowing of the nose, etc. Nasal catarrh may also be an accompaniment of other diseases, ulcers, outgrowths (Neubildung), caries, necrosis of the nasal bones. Inflammation from neighboring organs often spreads into the mucous membranes of the nose; thus, a very troublesome and intense coryza always accompanies a boil of the upper lip, and an abscess of the gum of the superior incisors. Nasal catarrh, too, is frequently a symptom of constitutional disease. The coryza which accompanies measles and exanthematic typhus belongs to this class, as do also the milder form of scarlatinous coryza, the coryza of congenital syphilis (see syphilis, vol. ii.), and probably also the nasal catarrh, which attacks a great number of persons during the prevalence of the influenza-epidemics, forming one of the symptoms of very general catarrh. In some respects, also, the iodine coryza, which constitutes one of the chief symptoms of iodic poisoning, comes in this category.

The somewhat common opinion, that a cold in the head is contagious, is contradicted by the experiments of *Friedrich*, who could not succeed in implanting the disease upon the mucous membranes of healthy persons by transferring to them secretions of persons suffering from catarrh in its several stages.

ANATOMICAL APPEARANCES.—At the commencement of an acute nasal catarrh, the capillaries of the nasal mucous membranes are surcharged with blood, the tissues are infiltrated, and the membrane, swollen by hyperæmia and œdema, gives out a colorless, thin, saline secretion. At a later period, while the hyperæmia and swelling of the mucous membrane diminish, the secretion becomes thicker and less transparent, from a copious admixture of young cells.

In chronic catarrh the nasal membrane is considerably swollen; it discharges a secretion, scanty in a few instances, but usually profuse and purulent, from the number of young cells which it contains. This often dries up within the nose into hard, dirty, dark-green crusts, and in some persons it evinces a great tendency to putrefaction without any assignable cause.

In many instances chronic nasal catarrh produces catarrhal ulcers, the cell-formation not confining itself to the surface, but spreading into the substance of the mucous membrane. They remain superficial, as a rule, but sometimes, particularly in scrofulous and cachectic subjects, they penetrate more deeply, and may destroy the perichondrium and periosteum, and give rise to caries and necrosis of the cartilages and bones of the nose. In the secretion from the ulcer, especially one which has caused a caries, and necrosis of the cartilage and bones, a foul decomposition is almost constantly going on, which produces an extremely bad odor, an occurrence only occasionally observed in the secretion of simple chronic nasal catarrh.

In other cases, chronic nasal catarrh gives rise to polypous outgrowths. According to *Rokitansky*, "the latter sometimes appear, first, as a diffuse thickening over a large surface of the mucous membrane, particularly on the turbinated bones, growing with an uneven surface, and developing wart-like protuberances and fold-like ridges. Sometimes the growths are more circumscribed, assuming a rounded, pedunculated, pyriform shape. They consist of a gelatinous growth from the matrix of connective tissue of the mucous membrane, and from its glands, which frequently degenerates into cysts. Gradually, the material of which the growths are constituted is converted into firm, fibrous tissue. They contract or block up the air-passages, and finally may become visible in the openings of the nostrils."

SYMPTOMS AND COURSE.—The symptoms of acute nasal catarrh may be presumed to be generally known; and everybody, probably, having had personal experience of them, they need but very brief notice here. The patient first complains of a feeling of dryness and of a more or less complete obstruction in one or both nostrils, which induces a very prejudicial inclination to blow the nose. From time to time, at short intervals, there arises a titillating or prickling sensation within the nostril, which usually precedes the complicated group of reflex symptoms known as sneezing, but which may also occur every now and then without being followed by the sneeze. This dryness of the nostril is soon succeeded by a very abundant secretion, a colorless, transparent watery liquid, of saltish taste, flowing almost incessantly from the nose, and sometimes producing excoriation of the upper lip. According to *Donders*, the irritating quality of the now strongly-alkaline secretion is due to the quantity of ammonia which it contains, while the proportion of chloride of sodium in it is less than its salt taste would lead one to suppose.

The senses of smell and taste become perverted, the tone of the voice is nasal. In almost all instances the catarrh extends into the mucous membranes of the frontal sinus, and, if the attack be severe, the patients complain of a feeling of pressure or of distressing pain in the forehead. We can perceive the redness and swelling of the mucous membrane as far as the eye is able to reach. In catarrhs of greater intensity, the redness and swelling extend from the mucous membranes to the skin of the nose and cheeks. A cold in the head is often complicated by a conjunctivitis. The patients avoid the light, and copious tears flow from their reddened eyes into the nostrils and over the cheeks. Should the fauces participate in the nasal catarrh, there is difficulty of swallowing; if the air-passages be implicated, cough and hoarseness accompany the symptoms. Propagation of the disease into the eustachian tube induces slight pain and roaring in the ears, or a temporary hardness of hearing.

The group of symptoms which we have just described is almost always accompanied by fever and general constitutional disturbance; particularly if the catarrh have acquired great intensity or be spread over a wide extent of surface. It may be slight in many persons, who are but little predisposed to febrile reaction; in others, who are usually spoken of as irritable persons, it is highly oppressive.

We have already depicted the symptoms of catarrhal fever, the often-recurring chill provoked by every change of temperature, the painful bruised sensation of the limbs, the loss of appetite, etc., while treating of bronchial catarrh.

The duration of acute nasal catarrh is usually short; indeed, the secretion generally becomes less profuse, and grows thicker and more opaque, even on the second or third day. It then loses its salt taste; the alkaline reaction is less marked; it takes on a yellowish or yellowish-green tinge, and dries, especially during the night, into hard crusts, which adhere firmly to the mucous membrane. The titillation in the nostrils and the sneezing are less frequent, the frontal headache abates, the swelling of the mucous membrane subsides, and, all liquid or inspissated secretion having been discharged, the nasal passages become free again. The febrile constitutional disturbance seldom lasts longer than a day or two. Those symptoms, too, which appertain to the spreading of the catarrh into the neighboring mucous membranes, generally begin to lose their intensity or even cease entirely toward the end of the first week; and in most cases the disease terminates in complete recovery on the fifth, sixth, or eighth day. It is very rarely, and only in scrofulous subjects, that a cold in the head becomes protracted and changes from an acute into a chronic affection.

While acute nasal catarrh is a complaint as common as it is harmless, it sometimes proves dangerous to infants at the breast, because the obstruction of their nasal passages, which are at all times narrow, makes it difficult for them to suck. If we do not feed with a spoon in such cases, life itself may be endangered in ill-nourished or feeble children.

In chronic nasal catarrh we do not usually find the feeling of prickling in the nose, the sneezing and the frontal pain, and the febrile general suffering. The swelling of the mucous membrane, however, creates a permanent narrowing of the nasal passages.

This disease owes its vulgar name of "snuffles," or "Stockschnupfen," to the more or less complete closure of one or both halves of the nose, by which inhalation of the air is impeded, and the voice acquires a nasal tone. The secretion of the diseased mucous membrane is sometimes purely mucous and sometimes muco-purulent, and its quantity varies. It is not always the more profuse and purulent secretion which shows the greatest tendency to putrefy. In fact, the so-called "*stinknase*," "*punaisie*," *ozæna*, is sometimes observed where the secretion is so scanty, even in chronic catarrh, that the existence of such catarrh has been overlooked, and it has been asserted that *punaisie* depends upon a fetid exhalation from the mucous membrane, and not upon the stench from putrid secretion. That the secretion should decompose more readily where the nostrils are unusually contracted is not improbable; and an analogous condition may, at all events, be adduced in young children who suffer from intertrigo behind the ears, in whom the secretion from the sore in the narrow cleft between the ear and the head is very apt to putrefy, and to emit a foul odor. If the secretion be copious and purulent, the blackish-green crusts, of stony hardness, above mentioned, frequently form, some of which are expelled by blowing the nose, while others are sucked into the pharynx through the posterior nares, and are then hawked up. In many instances we find the posterior wall of the pharynx covered with similar crusts. Chronic catarrh of the nose is an extremely obstinate disorder, often defying all treatment, and continuing for years, with varying intensity. It is often difficult, or even impossible, to determine whether the malady have led to ulceration and to ozæna, in the narrowest sense of the word. The very fact that the fetid odor of the secretion is not pathognomonic of ulceration of the nasal mucous membrane, and that it may be present also in simple chronic coryza, has made it necessary for those physicians who include all diseases of the nose, accompanied by an offensive discharge, under the term ozæna, to suppose the existence of two forms of ozæna, an ulcerous and a non-ulcerous form. It is only when the ulcers are situated so low down that we are able to see them by means of suitable dilatation and illumination of the nostrils, that a positive diagnosis is possible. Even the superficial ulcers of the nasal mucous membrane, which do not penetrate to the periosteum or perichondrium, are very refractory to treatment. Apart from the constantly-repeated provocations which they suffer upon blowing the nose, the close adhesion of the mucous membrane to the bone or nasal cartilages renders it almost impossible for the edges of the ulcers to approach one another, a condition which greatly impedes cicatrization. We shall speak of syphilitic ozæna in our second volume, where destruction of the nose by lupus, often called scrofulous ozæna, up to the point at which it passes into the province of surgery, is also to be discussed.

The question whether the nasal passages are obstructed by swelling and hypertrophy of the mucous membrane, or by a polypous growth, always remains doubtful until the polypus becomes accessible to sight or touch. In patients suffering from obstructive catarrh (*Stockschnupfen*) of one or both nostrils, particularly when the secretion is more or less covered by blood after violent blowing of the nose, we should never neglect exploring the cavities of the nose, both in front and rear, with the utmost care.

The method of examining large polypi and the symptomatology of such affections belong to the domain of surgery. I therefore pass by this subject, as well as that of the other growths in the nose, and the affections of its adjacent cavities, referring to the surgical text-books, and, above all, to the classical work of my colleague, *Bruns*.

TREATMENT.—Various abortive methods of treatment for acute nasal catarrh have been proposed, but none of them, neither swabbing the nostrils with sponges or charpie, nor syringing, nor touching the mucous membrane with a pencil dipped in solution of astringent or narcotic medicament, nor the application of narcotics in the form of powder, nor the inhalation of the vapor of vinegar, nor the so-called *dicta sicca*, have obtained general approval. The production of active diaphoresis, which has, indeed, in many instances, cut short acute nasal catarrh, is the only procedure worthy of adoption. Where the opportunity offers for a Russian bath, direct a patient, who is beginning to suffer from a cold in the head, to make use of one, observing all the precautions, upon which the success of this somewhat heroic procedure depends. In most cases, we should confine ourselves to advising the patient to keep his room for a day or two, and to swallow some hot drink from time to time, to keep the head and feet warm, not to use silken or cotton, but linen, pocket-handkerchiefs, and to change them frequently; to smear the upper lip with lip-salve, in order to protect it, by means of a film of grease, from the irritating action of the acrid secretion. The inhalation of warm vapor at the outset of a cold, while the nose is still dry, is advised by many. The snuffing of cold water into the nostril is not dangerous, as is vulgarly supposed, but it fails to afford a lasting relief, and sometimes even appears to protract the morbid process. In the latter stages of acute coryza, after the irritability of the mucous membrane has given place to a more torpid condition, a long walk in the fresh air, or even an occasional pinch of snuff, accelerates the cure. In infants at the breast, who are as yet unable to clear their nostrils, it is necessary to free them of the obstructing secretion by syringing out the nose with lukewarm water, and to feed them by the bottle, or with a teaspoon, as long as the obstacles to sucking continue.

In the treatment of chronic nasal catarrh, particular attention must be paid to any constitutional defect which may exist. The exhibition of cod-liver oil is indicated where there seems to be a scrofulous condition (presently to be described). In bloated, flabby subjects, with an over-tendency to obesity, a depletive treatment by the systematic exhibition of laxatives, as well as the cold-water cure, is suitable. Local treatment is of the utmost importance in the cure of chronic coryza. The most effective measure is pencilling the swollen mucous membrane with a solution of nitrate of silver (gr. iv—ʒ ss to ℥ j), or cauterization with the lunar caustic in substance, repeated from time to time. The local employment of mercurials, in the form of snuffs, is in great repute in such cases (calomel, hyd. precip. rub., āā gr. xij, sacch. alb. ℥ ss), as are also dilute solutions of corrosive sublimate injected into the nostrils. The preparations of alum, zinc, lead, tannin, etc., are less to be recommended. When the discharge is offensive, and should this condition not be corrected by the treatment above suggested, we may try whether the injection of weak solutions of chlorine, or of iodine or creasote (iodini puri, gr. ij—iv, potas. iodid. gr. iv—viij, aqua ℥ vj), will not give more satisfactory results.

Catarrhal ulcers require essentially the same treatment as chronic catarrh. Local treatment, however, especially the touching of the ulcers with caustic, is demanded where there is ulceration, even still more imperatively than in simple catarrh.

The management of polypous growths belongs to the province of the surgeon.

## CHAPTER II.

### HÆMORRHAGE FROM THE NASAL MUCOUS MEMBRANE—BLEEDING AT THE NOSE—EPISTAXIS.

Etiology.—Rupture of the capillaries of the nasal mucous membrane, from internal pressure, is of far more common occurrence than rupture of the capillaries of other mucous membranes, or of other organs.

Most people have never suffered from any spontaneous bleeding, save bleeding at the nose. One person may be more liable than another is to such hæmorrhage; but few have never suffered from it at all. Hence, as bleeding at the nose is the most common of all forms of spontaneous hæmorrhage, it is not surprising that, when a morbid tendency to spontaneous bleeding arises, it should be these very capillaries of the nasal mucous membrane which generally, and indeed almost always, give way. The morbid state of the capillary walls, which renders them apt to tear—the hæmorrhagic diathesis—therefore, is probably more or less diffused over the entire vascular system of the body; but it is in the nose alone, the organ whose vessels at all times evince a diminished power of resistance, that the nutritive disorder of the vascular wall suffices to occasion rupture from simple pressure of the blood.

Predisposition to nasal bleeding is, on the whole, far greater in youth than in more advanced life; but it rarely appears before the period of the second dentition, and does not occur in very young children. It is, moreover, the fragile constitutions, with slender bones, relaxed muscles, delicate skin, rather than big-boned, muscular persons, who are troubled by bleeding of the nose. The nutrition of the capillary walls is especially liable to suffer from exhausting diseases, whether acute or chronic, and we see epistaxis occur with striking frequence, as one of the symptoms of acute or chronic marasmus, in the course of typhus, of tedious intermitting fever, of the acute exanthemata, pleurisy, peritonitis (particularly that insidious inflammation within the abdomen, which originates in the cæcum or colon), and also in the course of tuberculosis, caries, etc.

The opinion here advanced, that the capillaries of the nose are more prone to rupture than those of other organs, is materially supported by the phenomenon that bleeding of the nose is seen nine times ere bleeding from other organs is seen once, in diseases which undoubtedly affect the condition of the entire body, and not the nasal mucous membrane alone. We must also mention that, in nearly all affections of the spleen, nasal hæmorrhage is a common symptom, and that among ancient physicians, and to this day among the people, repeated bleeding of the nose, especially if from the left nostril, is taken for an almost pathognomonic symptom of such diease. Affections of the spleen, however, occur so often in connection with exhausting maladies, and these maladies again, when uncomplicated by disease of that organ, so frequently show a tendency to epistaxis, that the genetic connection between nasal hæmorrhage and disease of the spleen remains a matter of doubt. This is true as regards hyperæmia, simple hypertrophy, and lardaceous degeneration of the spleen; and it is only when diseases of this viscus give rise to leuchæmia that we can consider it as demonstrated that bleeding at the nose depends immediately upon an affection of the spleen, or, at all events, that it is brought about by disorders of nutrition, to which disease of the latter organ gives rise.

The exciting causes of epistaxis are usually of so trifling a character as generally to escape detection. Hæmorrhage from the nose, indeed, sometimes occurs in consequence of blows, or other injuries sustained by that organ, and is a frequent concomitant of catarrh, ulceration, and the development of morbid growths in the nasal passages; but such hæmorrhages occurring in subjects exempt from morbid predisposition are rarely of great magnitude, and scarcely ever require any active treatment. On the other hand, in individuals afflicted by a morbid tendency to bleeding, the plethora arising after a full meal often gives rise to epistaxis. Sometimes the use of spirits, or of coffee, tea, or other hot drink, as well as violent bodily efforts, acute mental excitement, and other influences which excite the action of the heart, may have the same effect. In another series of cases, rupture of the capillaries is induced by some trifling obstacle to the outflow of the blood from the head; but, as we have already remarked, in predisposed individuals, the exciting cause of the bleeding is not generally determinable; and although theoretically we may classify the causes of nasal hæmorrhages into plethoric, fluxionary, and obstructive, it is often difficult to decide, in particular instances, to which of these three categories a case belongs.

The frequent occurrence of instances where patients with habitual epistaxis always bleed from one and the same nostril, from which nostril a brisk hæmorrhage may always be made to spring by thrusting any thing into it, while no such result is obtained by a similar procedure at the other nostril, makes it seem likely that bleedings of this kind proceed from dilatation of some small blood-vessel in the lower and anterior part of the nasal cavity. If we consider that the vascular net-work of the lower turbinated bones and their mucous membrane is extremely well developed, there being a vascular plexus there of some magnitude, containing both arteries and veins; and if we reflect that, in some persons, such hæmorrhages come on with suddenness and violence, a continuous jet of blood spirting all at once from the nose, there can hardly be any doubt (in spite of our lack of anatomical proof of the fact) that the source of the epistaxis, from which some people suffer upon every trifling occasion, consists in a varicose dilatation and thinning of one or more of these blood-vessels. The remarkable fact that very violent hæmorrhage can often be stanched by merely plugging the nose in front, and that it is not often necessary also to tampon the posterior nares, would likewise seem to indicate that the source of the bleeding lies low down and in front (*Seitz*).

Anatomical Appearances.—The bodies of persons who have died of epistaxis putrefy very rapidly, and upon autopsy exhibit signs of the most complete anæmia. In other respects, the results of *post-*

*mortem* examination are negative, that is, we neither can find patulous vessels as the source of the bleeding, nor can we, under the microscope, demonstrate any anatomical change in the condition of the vascular walls, to account for their liability to rupture.

SYMPTOMS AND COURSE.—In many instances the hæmorrhages are preceded by certain premonitory symptoms. These consist simply in the sensations which are peculiar to hyperæmia and swelling of the nasal mucous membrane, so that, for some time before the bleeding begins, the patients complain of stoppage of the nose, or of pressure in the region of the frontal sinus, or else there may be signs of fluxionary or obstructive hyperæmia of the brain, or general vascular plethora. In either case, soon after the establishment of the hæmorrhage, the prodromal symptoms usually abate, and, as they are often more distressing than the hæmorrhage itself, the latter is generally regarded as "critical."

The symptoms of the actual bleeding, when once it has set in, require no detailed description. Blood flows from both, or (as is more common) from one nostril, either in drops, or in a continuous stream of greater or less profusion. Should the hæmorrhage begin while the patient lies sleeping upon his back, the blood readily flows through the posterior nares into the pharynx. Part of it may enter the larynx and excite a cough, and the patient, upon awaking, fancies, to his great terror, that he has had a hæmorrhage from the lungs.

In other cases, the blood is swallowed, enters the stomach, and, when afterward voided by vomiting, may give rise to confusion with gastric hæmorrhage. The blood first lost has almost always a somewhat dark color, and shows great tendency to coagulate in the vessel into which it is received, or upon the lips, and even within the nose. This coagulation generally checks the bleeding in a short time, acting either as a spontaneous tampon, or by spreading from the effused blood to within the capillaries themselves. In other instances, however, in which the blood shows little tendency to coagulate from the first, or in which the tendency diminishes more and more, the hæmorrhage lasts longer, and exhausts the patient, particularly if he already be enfeebled prior to the loss of blood.

It is a matter of daily experience that, the longer an epistaxis lasts, the more obstinate it becomes; and often, after a duration of three or four days, it can only be subdued by means of the tampon. In some cases, it becomes apparent that loss of blood aggravates the hæmorrhagic diathesis more than any other debilitating cause, since, after an exhausting epistaxis of several days' duration, hæmorrhages from other mucous membranes and bleedings into the structure of the skin (petectia) supervene. In such cases of abundant and persistent bleeding from the nose, the visible mucous membranes, especially the lips and the conjunctiva, assume a marked pallor; the skin acquires a dirty-white, waxy hue; the patients are extremely prostrate, complain of pain in the head and back of the neck, of uneasiness, and of palpitation of the heart, and readily faint away; and, should it not be possible to arrest the bleeding, a task which, under such circumstances, is only to be accomplished by the most energetic interference, death from loss of blood may ensue.

TREATMENT.—A moderate epistaxis occurring in a vigorous person, particularly when preceded by symptoms which abate when the bleeding begins, may be left to itself, as it will, generally, soon cease spontaneously. If the hæmorrhage be more profuse, and begin to tell upon the patient, or if the latter be already in a depressed condition, so that we may dread evil consequences from even a slight loss of blood, we should warn the patient against aggravating the flow by frequent snuffing and wiping the nose, and should apply cold to the nose and forehead in the form of cold compresses, or else cause him cautiously to snuff cold water, containing a little vinegar or alum, into the nostrils. As long as the bleeding continues, we should also forbid all violent bodily motion, as also the use of coffee, tea, hot soup, and other heating substances, and advise that his drink be cool and acidulated. Should this treatment be unsuccessful—if the patient, in spite of it, become perceptibly weaker; should the blood begin to assume a brighter color, or coagulate slowly or incompletely, do not lose too much time in the trial of other styptics, as sulphate of zinc, creasote, liquor ferri sesquichlorat, but proceed at once to the simple tampon; or, if this be insufficient, to the double tamponade, by means of the canula of *Belloc*.

The application of ice to the testicles in men, and to the breasts in women; of dry or wet cups to the nape of the neck; ligation of the extremities; holding up the arms over the head—all procedures followed by decided success in some instances—are only to be resorted to as long as the bleeding still remains within limits which do not threaten danger. The same holds good of the internal administration of acids, of secale cornutum, and of gallic acid. The longer we delay the tamponade, not only will it be so much the harder to arrest the bleeding of the nose, but, as the hæmorrhagic diathesis increases with the duration of the bleeding (see above), other hæmorrhages are all the more apt to complicate the epistaxis, and against these we possess less certain remedies than the use of the tampon. I know of several cases in which a fatal result was almost indubitably owing to the too dilatory employment of the tamponade.

# DISEASES OF THE CIRCULATORY ORGANS.

## SECTION I.

## *DISEASES OF THE HEART.*

### CHAPTER I.

### HYPERTROPHY OF THE HEART.

ETIOLOGY.—The term hypertrophy of the heart is applied exclusively to a thickening of the cardiac wall, arising from an increase in the volume of its muscular tissue. It is of importance to observe a sharp distinction between this true hypertrophy of the heart and other enlargements of the cardiac wall, due to heterologous deposits, which may be called false hypertrophy, since the effect which the one disease has upon the circulation and the symptoms to which it consequently gives rise are entirely different from those of the other. If, as not unfrequently happens, the hypertrophied heart undergo degeneration, and if the genuine hypertrophy be converted into a spurious one, we find that the effects of the former disorder gradually subside, and that, if at length the degeneration preponderate over the hypertrophy, a group of symptoms arise which are almost directly the reverse of those which formerly existed. We know that the muscles of the heart, as well as those of the rest of the body, waste away and become atrophied from insufficient supply of nourishment, or in diseases attended by consumption; and we also know that a most abundant supply of nourishment has but little effect in augmenting the bulk of the muscles. On the other hand, it is a matter of daily remark that the external more visible muscles of the body undergo hypertrophy whenever they are subjected to constantly recurrent and vigorous contraction. The muscles of the blacksmith's arm, and of the leg of the mountaineer, are thus hypertrophied, and, as we have told in a previous section of the work, hypertrophy of the unnaturally-tasked muscles of respiration is one of the chief causes of the permanent expiratory condition of the thorax of emphysema. We have not as yet any satisfactory explanation of this fact; but an interesting discovery has been made, that a muscle, kept for some time in a state of tetanic contraction by the application of electricity, maintains an increase of its volume for several hours.

Upon analysis of the conditions under which hypertrophy of the heart arises, it will be found that most of them consist in disorders by which both the vigor and the frequence of the cardiac contractions are increased. (According to the measurements of *Bizot*, the thickness of the ventricular wall continues to increase until late in life [unless general marasmus arise], which is only ascribable to the constant exercise of the cardiac muscles.)

It is admitted by the physiologists that there must be some regulator of the action of the heart, which is still unknown to us, and by which the energy of the organ is adjusted to meet the wants of the system, and its activity increased as obstacles, which the heart has to overcome, become augmented. It is a matter of importance, then, to be able to demonstrate that hypertrophy of the heart occurs whenever the function of the organ is permanently or repeatedly overtasked, and when the resistance, which it should normally encounter, is increased. To prove this is easy:

1. Hypertrophy of the heart almost always accompanies abnormal enlargement of its cavity (*dilatation*). When the heart is dilated, its capacity is increased, and, as the organ cannot discharge its normal load without expenditure of a certain degree of force, the effort requisite for the expulsion of its abnormal increase of contents must be proportionately greater, even though the resistance at the orifices and in the arteries be normal.

When we come to study pericarditis, we shall learn that there is a form of hypertrophy of the heart which is purely the result of dilatation. The immediate effect of the infiltration of the cardiac wall, which takes place in this disease, is dilatation; very soon, however, this is followed by hypertrophy, although no fresh obstacle to the outflow from the heart has arisen meantime. The first result of defective closure of the valves of the heart is also dilatation; and it is not until afterward that hypertrophy develops in the portion of the heart immediately before the diseased valve, and which is caused by the greater effort now required to expel the increased amount of blood which the heart contains.

2. Hypertrophy of the heart accompanies stricture of its outlets, and contraction of the great vascular trunks. No detailed explanation

is needed to show that, under such circumstances, the resistance which the organ has to surmount must be augmented. Obstruction of the arterial outlets of the heart and insufficient calibre of the aorta, whether congenital or acquired, is accompanied by hypertrophy of the ventricles. Stenosis of the auriculo-ventricular orifices is attended by hypertrophy of the auricles.

3. Hypertrophy of the heart occurs in aneurism of the aorta and of the pulmonary artery. It is a fact in physics that the resistance encountered by a liquid, flowing through a tube, is increased if the tube be contracted or expanded suddenly. Now, whenever the great vessels which spring from the heart have undergone any considerable aneurismal dilatation, particularly if the dilatation be of the circumscribed form, both of these conditions exist in the vessels, and thus an additional tax is imposed upon the function of the heart.

4. Hypertrophy of the heart is an accompaniment of obstruction occurring in the range of the aortic current, or of the current of the pulmonary artery. The greater and more extensive this obstruction, so much the fuller must the aorta or pulmonary artery become, so much the more intense the strain upon their coats, and so much the greater the resistance which the heart has to surmount.

The great majority of obstacles to the circulation, which give rise to hypertrophy of the heart, are met with in the pulmonary circulation, and hence cause hypertrophy of the right side of the organ. In a previous section, a large number of diseases of the lungs and pleura have come under our notice, in which hypertrophy of the right heart has been shown to be not only a necessary consequence of anatomical changes brought about by the affections of the lungs, but was even counted as a symptom of these diseases themselves. In one case evacuation of the pulmonary arteries is impeded, a part of its efferent vessels, the capillaries of the air-vesicles, having perished through emphysema. We have seen, too, how the capillaries are obliterated from the shrunken tissues of a cirrhosed lung; and that, in pleurisy, sometimes one-half of the vessels into which the pulmonary artery should discharge its contents are compressed and have become impervious. But strain upon the pulmonary artery is not dependent upon diminution of the number of its efferent channels alone. A mere impediment of the outflow of blood from the capillaries into the veins will produce the same effect; hence, to the catalogue of pulmonary diseases above mentioned, we must add that of the affections of the left side of the heart, which give rise to obstruction in the pulmonary vein. In treating of valvular deficiency of the left auriculo-ventricular passage, we shall consider the subject of "compensating" hypertrophy of the right ventricle more in detail.

Disorders of the greater (aortic) circulation, which give rise to hypertrophy of the left ventricle, are more rare. This is due to the fact that, in its vast system of vessels, the obliteration of a very great number of capillaries, and even the ligation and compression of the great vascular trunks, is compensated for by dilatation of the vessels of other regions. The impediment to the aortic circulation which most commonly embarrasses the action of the left side of the heart, and to which most cases of hypertrophy of the left side are commonly due (when they are not the result of disease of the aortic valves), is a degeneration of the arterial walls, generally known as "atheroma in its widest sense," and which we shall by-and-by describe in detail as *endarteritis deformans.* In this affection, which is often widely diffused throughout the wall of the arteries, the vessels not only become elongated and tortuous, so that the friction of the blood against the sides of the arteries is increased, but the elasticity of the arterial tunics (a most important auxiliary element of the circulation) is seriously diminished. In the obliteration of the aorta, which sometimes occurs close below the point of entrance of the ductus botalli (see Section III., Chapter IV.), the left ventricle is also found in a state of considerable hypertrophy. Occupations which require violent muscular exertion have likewise been enumerated among the causes of this disease. If this be true, such hypertrophy also belongs in this category. The number and size of the efferent vessels of the aorta are reduced by the pressure to which the capillaries within the contracted muscles are subjected, and thus the contents of the aorta and the tension of its coats are increased. *Traube* has shown by experiment, that an augmentation of pressure takes place in the aortic system during general contraction of the muscles. *Traube* finally counts the hypertrophies usually found on the left side of the heart, which (even according to *Bright*) often complicate the third stage of *Bright's* disease, as among those which proceed from disturbance of the aortic circulation. He supposes that an increase of tension within the aorta arises, partly from atrophy of the vessels, partly from abnormal fulness of the aorta, from which a reduced amount of liquid is withdrawn into the kidneys, which, by augmenting the obstacles to the evacuation of the left ventricle, give rise to hypertrophy. *Bamberger* brings forward serious objections to this explanation. He shows that the hypertrophy begins in the earlier stages of *Bright's* disease, that it does not affect the left heart alone, but often involves the whole organ; that we sometimes find the aorta not only undilated, but even narrowed, that it is improbable, and that there is no precedent for the supposition that the destruction of a few renal capillaries should produce a hypertrophy of the heart, to which the ligation of large arterial trunks cannot give rise.

5. Hypertrophy accompanies general plethora. It is easy to comprehend that, if the vascular system at large be overfilled, the obstacles which the heart has to overcome must be increased. It may, however, very properly be questioned, whether a permanent and general increase of the contents of the vascular system be possible. Such a condition would immediately be compensated for by augmented secretion, especially from the kidneys, as urine always continues to form as long as lateral pressure upon the renal arteries and vascular tufts of *Malpighi* is kept up.

A transient plethora, however, undoubtedly arises both after every hearty meal and after copious drinking. Persons who, by immoderate eating and drinking, often bring upon themselves this merely transitory plethora, persons who lead a gluttonous life (for example, travelling wine-sellers, who often eat and drink all day long), furnish no inconsiderable contingent to the general mass of cardiac hypertrophy.

In all cases, mentioned hitherto, it has been more or less distinctly demonstrable that the augmented action of the heart, which gives rise to hypertrophy, proceeds from increased resistance and from the requirements of the general organism for such increase of action. To this class of cases another must be added, in which hypertrophy is a result of increased cardiac action without increased resistance. The action of the heart is accelerated by excitement of the passions. In many persons we are forced to assume the existence of an exalted irritability, an erythism of the nervous system, particularly of the nerves of the heart, so that trifling causes serve to excite and strengthen its action. The use of strong coffee, tea, and spirits, has a similar effect, thus furnishing a fresh exciting cause to the class of hypertrophies treated of under our fifth heading. Such agencies, however, are far less productive of hypertrophy than those previously mentioned.

Finally, it must be admitted that we are ignorant of the pathogeny of quite a large number of cases of hypertrophy of the heart, and (in accordance with the objections of *Bamberger* to the views of *Traube*) we must reckon among these the hypertrophies which so often complicate *Bright's* disease without the coexistence of any valvular lesion, or other structural changes in the heart or blood-vessels.

We sometimes notice that cardiac hypertrophy confines itself to one side of the heart, or even to one ventricle or auricle. It can nearly always be shown that the cavity, whose walls are hypertrophied, has an unusual task to perform. It is more common, however, even though but one orifice be contracted, for the hypertrophy to extend more or less over the entire heart. The former condition is probably the more difficult of explanation, when we reflect that part of the muscular fibres of one ventricle pass over to the other.

ANATOMICAL APPEARANCES.—The weight of the normal heart amounts in adult males to about ten ounces, in females to about eight. A hypertrophied heart may weigh from one to two pounds. According to *Bizat*, the thickness of the left ventricular wall is computed, in the male at five, in the female at four and a half lines; the thickness of the right ventricle in males being almost two lines, in females one and two-thirds lines. The right auricular wall is one line in thickness, the left one and a half lines. Hypertrophy of the right ventricle may be considered to commence when the thickness attains six lines in the male, and five in the female. The right ventricle is hypertrophied if its thickness amount to three lines in the male, or two and a half in the female. In the most extreme cases of hypertrophy, the left ventricular wall may acquire a thickness of an inch or an inch and a half; and the right may be six or nine lines in thickness, while that of the auricles may amount to two lines, or in the left auricle even three lines.

This increase in volume sometimes is most marked in the fleshy wall proper, sometimes in the trabeculæ and papillary muscles. The former is most often found upon the left ventricle, the latter in the right ventricle. Hypertrophy may be *total*, that is to say, extending all over the heart, or *partial*, that is, limited to certain portions of it. We distinguish three forms of hypertrophy, according to the capacity of the hypertrophied portion—where the capacity is normal, *simple hypertrophy;* where the cavity of the heart is enlarged, *excentric hypertrophy;* where it is diminished, *concentric hypertrophy.* In the first and second forms, the size of the heart is increased; in the third, if the diminution of the cavity exceed the hypertrophy of the wall, the organ actually may be smaller than is natural.

Simple hypertrophy is not common. In many cases in which the hypertrophied heart seems to have its normal capacity, its cavity has been dilated during life, but has contracted energetically during the agony of death, so as to cause the dilatation to disappear in the cadaver. This form is confined to the left heart, particularly to cases in which hypertrophy of the heart complicates Bright's disease.

Excentric hypertrophy is the most common form, and is often met with extending over the entire heart, and should the hypertrophy and dilatation attain any great degree of magnitude, it may occasion the establishment of an "*enormitas cordis*," of a "*cor taurinum.*" In some cases excentric hypertrophy is restricted more to the left side of the heart; in others, to the right. In the former case, the capacity of the right ventricle often suffers, as the septum, the muscles of which belong, in great part, to the left ventricle, is made to project into the cavity of the right ventricle.

Concentric hypertrophy is exceedingly rare, although a normal heart, which has contracted strongly at the moment of death, has often been mistaken by inexperienced persons for such a condition. The very existence of this form has been doubted by *Cruveilhier*. *Rokitansky* and *Bamberger* express their opinion that, although rare, it sometimes occurs.

The shape of a heart in a state of general hypertrophy is that of an obtuse-angled triangle. If the hypertrophy be limited to the left side, the organ is usually longer, and has a more conical form. The lower end of the right side does not extend as far downward toward the apex as it otherwise should. In excentric hypertrophy of the right ventricle, the heart grows broader, and assumes a more spherical form. The right ventricle lies farther forward; the left is, as it were, pushed away from the thoracic wall. The apex is often small, consisting principally of the right ventricle.

The heavier the heart becomes, so much the deeper does it lie. The diaphragm is pressed downward, and the heart generally inclines more to the left side of the thorax. When the hypertrophy is excessive, and accompanied by dilatation, the base always keeps sinking deeper, and the organ assumes a transverse attitude, the base toward the right, the apex toward the left. When the right ventricle alone is affected, the heart projects more into the right side of the thorax. If the left alone is involved, the prominence is rather into the left side of the thorax. Hypertrophy of the heart depends, probably, upon a multiplication of the muscular fibres and primitive fasciculi of which its walls are formed, as *Foerster* has never been able to demonstrate the existence of any "thickening" in them.*

The color of the substance of the heart is a dark, brownish red. The consistence is often considerably increased, so that the walls of a hypertrophied heart do not collapse when cut open, as they otherwise should do.

SYMPTOMS AND COURSE.—It is difficult to furnish a picture of pure hypertrophy of the heart, since this affection, as we have seen, scarcely ever exists independently, but is almost always an accompaniment of other grave diseases of the organ, or of the great vessels, etc. In fact, these complications often completely neutralize the effect which the hypertrophy would have, were it to exist alone. A great number of the phenomena set down as symptoms of cardiac hypertrophy certainly are not dependent upon that cause; and indeed would exist in a much more marked degree were it not for the coexistence of the hypertrophy of the heart with the main disease. This is especially true of cyanosis and dropsy. Often as we see these symptoms in such cases, they are always to be ascribed to the complications, and never to the disease itself, if the hypertrophy be genuine; that is, if it depend upon multiplication of the normal muscular fibres of the heart. *Bouillaud* justly denounces the statements of authors, according to whom a hypertrophy of the heart gives rise to cyanosis and dropsy. He says: "Can any one, according to sound physiology, suppose that a true and simple hypertrophy of the heart can be capable, by itself, of producing phenomena which indicate embarrassment and weakening of the function of the heart?" In spite of this clear and vigorous protest, cyanosis and dropsy are still set down among the symptoms of hypertrophy of the heart.

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* *Rokitansky* and *Bamberger* certainly speak of an increase in bulk of the primitive muscular bundles.

Let us first suppose a hypertrophy of both ventricles. The blood must then be propelled into the arteries with unwonted energy upon every stroke of the systole, and if (as is usually the case) the ventricles be at the same time dilated, the arteries will become abnormally full. But just as, during the systole, the hypertrophied ventricle completely expels its contents, so during diastole the efflux of the blood to the heart from the veins must be made easier. The veins become more empty, while the arteries fill. Nor can the capillaries ever undergo any undue distention, for the outflow from them becomes easy in proportion as the *vis a tergo* is increased. The effect, then, of a general hypertrophy of the heart is, *that the arteries become fuller, the veins less full, and that the circulation is accelerated.*

If the left side alone be hypertrophied, its contents must be more completely expelled than if its walls possessed merely their normal thickness. Hence (and all the more so if dilatation also exist) the aortic system becomes over-filled, while the volume of blood in the pulmonary system must be correspondingly reduced. In spite, however, of the over-filling of the aortic system, it can never become so great as to cause dropsy or cyanosis by over-distention of the capillaries and veins. This is prevented by the depletion which takes place in the vessels of the pulmonary circulation. The vessels of the lung, being imperfectly filled, offer but little resistance to the entrance into them of blood from the right ventricle, which, although not hypertrophied, can propel its blood with unusual ease. Thus the engorgement of the vena cava subsides, its blood, under the increased pressure from behind, flowing readily into the empty right heart; and the right ventricle, being well supplied with blood, and its contents readily passing out into the meagrely-filled vessels, soon is propelling quite as much blood as the left ventricle, into which the blood runs, under a reduced pressure, and which discharges its contents into the over-filled aorta with difficulty. Hence the effect of hypertrophy, especially of excentric hypertrophy of the left ventricle, is to produce an abnormal fulness and engorgement of the vessels of the aortic circulation, which does not extend into the veins (the outflow from which, indeed, is facilitated), to diminish the fulness of the vessels of the smaller circulation, and to accelerate the current in both systems. The latter fact is easy to account for, if we remember that both ventricles set an abnormally large amount of blood in motion with every systole; the left, because it is hypertrophied, the right, because it sends its blood through scantily-filled vessels.

Should the right ventricle alone be hypertrophied, then, conversely, the volume of blood in the lesser circuit is increased, and that of the greater is diminished. But here, no sooner does the right ventricle discharge more blood than the left, than the flow from the right ventricle into the over-distended pulmonary artery becomes embarrassed, while the flow from the unhypertrophied left ventricle into the scantily-charged aorta is rendered easier. Upon the other side the blood pours into the left heart from heavily-charged veins, and into the right from veins which are imperfectly filled, so that here, too, both ventricles soon begin to propel an equal amount of blood, without which the whole of the blood would collect in the pulmonary system. Hence, hypertrophy of the right ventricle would result in augmentation of the contents of the pulmonary system, reduction of that of the aortic system, acceleration of the circulation, with easier outflow from the pulmonary veins than from the vena cava.

From the foregoing, in which we have mainly adopted the lucid analysis of *Frey*, it is easy to perceive what symptoms hypertrophy of the heart occasions, and what variations must arise, according as the entire heart is involved, or portions of it. We are treating now, however, of uncomplicated hypertrophy, where there is no obstacle to the current of the blood, the effects of which, hypertrophy would tend to counteract, and can only consider the subject of consecutive hypertrophy when we discuss the subject of valvular diseases, the symptoms of which they modify.

Total excentric hypertrophy of the heart is the form which most frequently arises without complication. In most instances, persons thus affected feel perfectly well, and it often happens that the existence of the malady is not detected until the physician makes a physical exploration of the chest, after the occurrence of an apoplectic stroke, or that it is first observed *post mortem*, after an apoplexy which has cost the patient his life. The patient has had no occasion to consult the doctor, the doctor none to examine the chest. Thus it is with the majority of cases, which, though really under observation, are not understood. The pulse of such patients is full and strong, the carotids pulsate visibly; in all the greater arteries we hear, during the systole of the ventricle, a distinct ring. The face is reddened, the eye glitters, and sometimes is remarkably prominent. The functions are normal. Respiration, as long as the heart has not attained an excessive development, is not materially embarrassed in pure hypertrophy. However, when a "*cor bovinum*" displaces the lungs to either side, and depresses the diaphragm, there may be a sensation of fulness in the chest, of pressure in the epigastrium, and often a considerable degree of shortness of breath. In many instances, the patients complain of palpitation of the heart, particularly when excited, although these are not, by any means, constant signs. We must often wonder that the impulse of a heart, strong enough to jar the chest like the blow of a hammer, should cause the patient so little inconvenience, or afford absolutely no subjective symptoms whatever.

In the course of total excentric hypertrophy, especially if any particular cause excite the action of the heart, symptoms of active hyperæmia and fluxions arise in those organs whose vascular walls, possessing but a feeble power of resistance, are liable to an increased afflux of blood into them whenever pressure of the whole arterial system is augmented, as in the brain and the bronchi. In consequence of the fluxion to the brain, if the patient run, make use of heating drink, or undergo any physical excitement, headaches arise, or spots before the eyes, buzzing in the ears, dizziness, formication, etc., and fluxion to the bronchial arteries occasions swelling of the bronchial mucous membranes, wide-spread sibilant rhonchi, great dyspnœa, attacks of asthma, which often soon subside after the patient has lost a little blood, or after the administration of a cathartic, which, by relieving pressure upon the abdominal arteries, facilitates the circulation in the thoracic and abdominal aorta. These attacks must not be attributed to hyperæmia in the region of the pulmonary artery. The symptoms, especially the wide-spread cooing in the chest, and the character of the dyspnœa, which strikingly resembles that of a bronchial asthma, show distinctly that the fluxion has taken place in the province of the bronchial arteries.

It is by no means rare for cerebral apoplexy to occur in a case of total excentric hypertrophy; indeed, we shall see that, in the majority of cases of ruptured blood-vessels of the brain which have been observed in young subjects, hypertrophy of the heart, either total or left-sided, was the assignable cause. The frequence of apoplexies of the brain is in part due to the fact that the vessels of the brain are thinner than those of other organs, and hence are more liable to rupture when unduly distended; and in part, also, to the circumstance that the coats of the arteries in hypertrophy of the heart are often atheromatous, and hence give way easily. That there is a genetic connection between atheroma of the arteries and cardiac hypertrophy (which *Rokitansky* and *Virchow* also admit) can hardly be longer called in question, after the observations of *Dittrich*, according to whom the pulmonary artery, which is otherwise rarely the seat of atheroma, is often found to be atheromatous where there is a hypertrophy of the right side of the heart. Should the patient survive the first or second attack of apoplexy, he may attain a tolerably advanced age. In other cases the hypertrophied heart degenerates, when the malady assumes a very different aspect. Venous obstruction, dropsy, etc., arise, symptoms which we shall discuss more in detail in treating of degeneration of the substance of the heart.

The symptoms of total, simple, uncomplicated, excentric hypertrophy of the left side of the heart, which is next in frequence of occurrence, must, of course, closely resemble those of total hypertrophy. Here, too, the circulation is accelerated, the arteries are extremely full, and there is no engorgement of the veins or capillaries, the blood flowing into the right side of the heart with ease and rapidity. The respiration is not injuriously affected by diminution of the contents of the pulmonary system, as the negative influence exerted upon oxygenation by emptiness of the vessels is fully counterbalanced by the positive one of acceleration of the circulation. Here, too, we seldom hear any complaint from the patient. The pulse is full and strong, the complexion healthy, and the functions normal. Derangement of the respiration is even still more rare in this form of disease than in total hypertrophy of the heart, as, in these cases, the heart seldom encroaches upon the cavity of the thorax. Palpitation is a frequent, but by no means constant, symptom. The malady usually terminates in apoplexy.

Often as the right ventricle takes part in an excentric hypertrophy of the left ventricle, and extremely often as hypertrophy of the right side of the heart accompanies derangements in the pulmonary circulation, and lesions of the valves of the heart, yet simple hypertrophy is extremely rare in the right ventricle, indeed it is questionable whether it ever has been seen. Any description of simple hypertrophy of the right side of the heart which we might make, must be a fictitious one, not based upon actual observation. At all events, intense dyspnœa and œdema of the lung, which have been set down among its symptoms, are as little dependent upon hypertrophy of the right ventricle as are cyanosis and dropsy caused by hypertrophy of the left; indeed, as we shall see, hypertrophy actually diminishes the dyspnœa which the main disease has occasioned, just as hypertrophy of the left side, when complicating valvular derangement, long averts the occurrence of cyanosis and dropsy.

With regard to concentric hypertrophy, from the great rarity of this form of the disease, the very existence of which has been doubted by authors of merit, we have no available clinical data whereon to base an account of its symptomatology. If the capacity of a concentrically hypertrophied heart be considerably reduced, symptoms essentially different from those hitherto described must arise. In spite of the increase of muscular development, the quantity of blood thrown into the arteries can only be small; the outflow from the veins into the narrowed heart must be impeded, so that cyanosis and dropsy may ensue.

*Physical Signs.*—In young subjects suffering from excentric hypertrophy of considerable extent, we may sometimes observe a distinct prominence over the region of the heart, which is not to be confounded with the deformity which proceeds from rachitis. In older persons, whose costal cartilages have become ossified, this symptom is not met with even in cases of "*enormitas cordis.*" Besides this, the shock of the heart is observed to extend widely over the thorax, and is visible at unusual places. We shall consider this more attentively while speaking of palpation.

*Palpation.*—In the majority of healthy persons, we see and feel, during systole of the ventricle, that the spot in the thoracic wall corresponding to the apex of the heart receives a concussion, is shaken, and that a limited space between the two adjacent ribs is then made to bulge forward. This phenomenon, the impulse of the heart, arises from the force with which the heart is depressed and pressed against the thoracic wall when the ventricle contracts. Although the views of the different investigators may vary as to the cause of the systolic descent of the heart, one side asserting it to be due to elongation, and stretching of the great vessels; another that it depends upon the recoil which the heart makes when the blood is forcibly expelled from it, somewhat like that of an exploding gun; yet all agree that the heart descends during the systole. Now, if we bear in mind that the heart does not hang free in the chest, but lies upon the diaphragm, a surface which slopes forward, it will become apparent that the heart, when pressed downward, must also be pushed forward against the wall of the chest. If the apex of the heart then strike upon an intercostal space, it is driven into it, and causes it to bulge. If, however, it meet a rib, or if the intercostal spaces be too narrow to admit the apex between the adjoining ribs, then, instead of the impulse of the apex, a feeble circumscribed shock is felt upon the ribs or intercostal spaces. It is very plain, that the beat of the apex will be most often visible in persons who have wide intercostal spaces, and the point of whose heart inclines more outwardly; whereas the circumscribed concussion is more commonly felt in individuals with narrow spaces, and whose apices point more inwardly. Even in the healthy subject, if the heart be stimulated into more vigorous action, a feeble jar may be noticed, not only over the apex, but throughout all that part of the chest which comes into contact with the heart. So, too, when the heart is excited, other conditions being normal, a shock may be observed in the epigastrium, which, however, is not to be confounded with the so-called *pulsatio epigastrica.* This epigastric pulsation is produced by the left lobe of the liver, which is driven downward a little by every systolic movement.

In hypertrophy of the heart, various deviations from this normal cardiac impulse are met with. Very great intensity of impulse is almost peculiar to hypertrophy, while a less violent pulsation may arise from mere excited action of a heart of normal size. *Skoda* recognizes two degrees of abnormal intensity in the beat of the heart, one in which the head of the auscultator, when laid upon the chest of the patient, perceives a strong, jarring sensation, but in which the thoracic wall and the head of the listener are not lifted by the shock; the other, in which the thoracic wall is distinctly elevated during the systole, and sinks again with the diastole. Here, too, if this lifting of the thoracic wall take place rapidly, a shock is imparted to the head. It is this second degree, this distinctly heaving heart-shock, which is pathognomonic of hypertrophy, and which does not occur in any other disease. (This heaving cardiac impulse, however, must extend over a large area of the thoracic wall, to warrant a certain diagnosis of hypertrophy; as the apex of a normal heart, when it beats upon an intercostal space, will produce distinct elevation of the point of impact, and will lift the finger when laid upon it.) The jarring impulse, if a constant and not merely a transitory symptom, is also decidedly indicative of hypertrophy. As a rule, the beat of a healthy heart is only felt over a spot covering one or two intercostal spaces, while the shock caused by the organ when hypertrophied is often perceptible over a region including several of these spaces. In total excentric hypertrophy it is diffused both longitudinally and transversely. In hypertrophy of the left ventricle the heaving pulsation is most distinct at the apex, and thence spreads somewhat longitudinally, less so in the transverse direction of the organ.

In excentric hypertrophy of the right side of the heart, the thoracic wall between the apex and the lower edge of the sternum, or even the sternum itself, is shaken. All these variations are attributable to displacement of the lung, and to the more perfect contact thus existing between the heart and the parietes of the chest. Observation of the beat of a hypertrophied heart, moreover, reveals a displacement of its apex. Under normal conditions the apex almost always beats at the fifth intercostal space, and it is only when the spaces are very wide, or when the abdomen is distended, that the apex beats at the fourth space, and when the spaces are narrow, at the sixth space. According to *Seitz*, it is more often felt in the fourth than in the fifth space in children. The point in the fifth intercostal space, at which the apex is usually felt, is from half an inch to an inch below the nipple, at the parasternal line (that is, a vertical line running midway between the nipple and the left border of the sternum). Sometimes the beat is a little without this line, more rarely somewhat within it. If the heart be considerably enlarged, the impulse is not exclusively visible at the apex, as the chest suffers a jar from contact with other portions of the organ. Hence, we must know how to find the apex. The rule is, to assume that the lowest and most external point at which the impulse is distinctly felt corresponds to the apex. In all forms of excentric hypertrophy—the total form, as well as that of either side—the apex may be displaced to the left, and in total or left-sided hypertrophy it may likewise move farther downward, and be found in the sixth or even the seventh intercostal space. This very seldom happens when the right side is affected, and only when the right ventricle projects beyond the apex. With a little care and practice it is easy to distinguish the sensation arising from pressure of the heart against the thoracic wall (which is the sole guide for estimating the extension of the cardiac impulse) from that caused by participation of the surrounding region in the shock.

*Percussion.*—The normal region of dulness over the heart forms a triangle which is bounded inwardly by the left border of the sternum, from the fourth rib downward; externally by an imaginary line drawn from the sternal edge of the fourth rib to the point at which the apex beats. Below, the dulness is usually merged in that of the left lobe of the liver, and it is only in cases where the latter extends less to the left than usual that the cardiac dulness is bounded on the lower side by the sixth rib or seventh intercostal space.

This normal dulness of the cardiac region becomes greater in excentric hypertrophy of the heart. In hypertrophy of the left side it becomes longer, in that of the right broader, and in total hypertrophy it is increased both in the vertical and transverse directions, i. e., is both longer and broader. In hypertrophy of the left ventricle, wherein the cardiac dulness extends downward rather than upward, observation of the situation of the heart-stroke is of more moment, in diagnosis, than percussion, which here often fails.

Besides the cardiac dulness, that is, the dulness upon percussion arising from contact of the compact heart with the wall of the chest, many authorities also describe a region of flatness, which is found where a thin layer of lung lies between the heart and the thoracic wall, and indeed a dispute has arisen between them, as to which of these percussion-signs is entitled to the name of cardiac dulness, and which that of cardiac flatness. Such niceties and disputes over names are of no practical use. They neither benefit the patient nor prómote the progress of science.

*Auscultation.*—We follow *Bamberger's* account of the normal sounds of the heart. According to him, the first sound, that which is heard simultaneously with the heart-stroke, and which corresponds with the systole of the ventricle, is produced both in the ventricles and in the arteries: in the ventricles, by the sonorous vibrations into which the tricuspid and mitral valves are thrown, when placed in a state of tension by the blood as it is forcibly expelled; in the pulmonary artery and aorta, by the sound created by the distention and stretching of the walls of those vessels by the passage of the blood-wave. We are forced to the latter supposition, by the circumstance that we can hear a systolic sound in all the larger arteries, even at a distance from the heart, which could not possibly be propagated from the ventricles, whenever the vessels are much distended. The second heart-sound, which is heard during the diastole of the ventricle, and which is separated from the previous one by a short pause, and from the sound which follows by a more extended interval, is produced in the arteries alone. Although it is audible in the region of the heart, yet it is conducted thither, as no sound can well arise in the heart itself during its diastole. Its source is in the arteries, where it is created by the flapping of the semilunar valves, which are put on the stretch by the diastole, and receive a shock from the blood which is driven against them. The heart-sounds are never altered nor converted into murmurs by simple hypertrophy. On the contrary, when the heart is hypertrophied, the sounds are more distinct and louder, the mitral and tricuspid valves being exposed to heavier concussion and thrown into stronger vibration, the aorta and pulmonary artery being fuller, and hence vibrating more actively, owing to the additional flow of blood which they receive, and to the greater shock falling upon the semilunar valves from increase of the arterial contents. When there is much hypertrophy, a peculiar metallic sound is audible (cliquetis métallique) during systole, which appears to come from vibration of the thorax.

DIAGNOSIS.—Not only are the subjective signs of hypertrophy of the heart, and their concomitant derangements of the circulation, liable to be overlooked, but even the physical signs may fail to reveal the existence of the malady. When the left lung is emphysematous, and intervenes between an enlarged heart and the thoracic wall, there is often no abnormal increase of the cardiac impulse, which, indeed, may actually become diminished in strength, or even be quite imperceptible. In similar manner, the cardiac dulness may be reduced in area, rather than increased, and even the heart-sounds themselves, when muffled by an emphysematous lung, may fall feebly upon the ear.

The following are the diagnostic points deduced by grouping the subjective and objective signs of excentric hypertrophy of the left ventricle: Visible pulsation of the carotids, loud systolic sound in the larger arteries, and a full pulse, visible even in the smaller arteries; an abnormally strong heart-stroke, extending over the length of the heart; a depression of the apex, extension of the cardiac dulness, intensification of the heart-sounds in the left ventricle and in the aorta, and sometimes a metallic click.

Excentric hypertrophy of the right heart declares itself by the following objective signs: Augmented heart-stroke, which often extends along the sternum and the left lobe of the liver; dislocation of the apex, which extends outward, but hardly ever downward; extension in width of the cardiac dulness, intensification of the cardiac sounds in the right ventricle, and in the pulmonary artery. The difference in the strength of the heart-sounds is most distinctly perceptible in the arteries, and particularly in the second sound, so that a stronger second sound from the pulmonary artery, which is easily recognizable, even in extreme emphysema, is a most important token of hypertrophy of the right side of the heart.

The sum of the objective symptoms of hypertrophy of either side of the heart furnishes the physical signs of total hypertrophy. The arteries and the pulse make the same manifestations as in hypertrophy of the left side; the heart-stroke is considerably stronger, extending both longitudinally and transversely; the apex is situated low down, and far to the left; the cardiac dulness is extended in all directions, and all the heart-sounds are louder.

As it is of importance, in diagnosticating the several forms of this disease, to be able to compare the sounds which are audible at the origins of the arteries and auriculo-ventricular orifices, we must obtain an exact knowledge of the points in the thorax which correspond to the arterial and venous openings, or at which, at all events, each sound may be most distinctly heard and isolated from the others. The rule here is to seek for the aortic sounds at the right edge of the sternum, at the level of the third costal cartilage. This sound, it is true, is usually more audible upon the left of the sternum than upon the right, but at the left side, where the pulmonary artery lies directly over the aorta, it is often difficult to determine whether a sound proceeds from the aorta or pulmonary artery. For the sound of the pulmonary artery we listen at the middle of the third costal cartilage; for that of the tricuspid, at the lower end of the sternum, on a level with the fourth intercostal space.

The mitral sounds are less distinctly audible at the spot in the thorax corresponding to the situation of the mitral valve, but can be heard and isolated better at the region of the apex, which lies about in the third intercostal space, an inch and a half from the left border of the sternum. This is owing to the fact that the mitral valve is separated from the anterior wall of the chest by the right side of the heart, and from the lateral wall by the lung. These media are poorly adapted for the transmission of the sounds of the mitral to the ear, or for their isolation from the sounds arising in the right side of the heart. On the other hand, the apex, which belongs to the left ventricle alone, and which lies immediately in contact with the wall of the thorax, is well calculated to isolate the tones of the mitral, and to conduct them to the ear.

The sounds and murmurs which arise in the various orifices of the heart are often heard, with greater distinctness, at other points than those just mentioned. This is sometimes owing to an elevated or depressed attitude of the diaphragm, or to displacement of the mediastinum, and sometimes, indeed, no reason for the variation can be assigned. In order not to be led into error of diagnosis by such irregularities in determining the source of a murmur, we must not attach too much diagnostic importance to the situation of the point where the sound is most plainly audible, but should rather rely upon the concomitant signs of enlargement of one or other portion of the heart.

To save repetition, we shall defer the discussion of the differential diagnosis of hypertrophy and dilatation of the heart, and of pericardial exudation, etc., until we shall have learned the symptoms of the latter diseases.

Prognosis.—Of all diseases of the heart, hypertrophy admits of the best prognosis, if we accept the narrowest meaning of the term which we have assigned to it. In many instances, in which we shall find hypertrophy as a complication of other diseases of the heart, the compensatory hypertrophy actually mitigates the danger of the chief disease.

Patients with simple hypertrophy of the heart may live to a great age. If they die early, death is generally due to hæmorrhagic effusions into the brain or lungs, for the prevention of which occurrences a careful treatment and a judicious regimen do not seem to be without efficacy. The prognosis, however, is often rendered more grave by the occurrence of a consecutive degeneration of the substance of the heart. With the transition from genuine to spurious hypertrophy, the picture changes, and many dangers arise.

TREATMENT.—It is certainly out of our power to cure a hypertrophied heart by any mode of treatment, although we do not deny that such a heart may undergo atrophy quite as well as a normal one. Much, however, may be accomplished in the way of checking the advance of the disease, and in moderating the dangers to which it gives rise. We are not now referring, of course, to the cases in which hypertrophy is a complication of another malady of the heart or lungs, but to that form of the disease which exists in a certain degree independently, such as we see in drunkards, gluttons, etc. Here the disease generally remains unrecognized, until the enlargement of the heart begins to encroach upon the lungs, or until dizziness, muscæ volitantes, or other symptoms of fluxion to the brain, arise, or, what is still more common, until an apoplectic attack calls attention to the state of the organ. If we now give our orders with decision, we may reckon upon punctual obedience upon the part of the patient, who previously would scarcely have paid them any attention, but to whom the inconveniences just alluded to become a source of the greatest solicitude. Pathogeny makes the treatment clear. Such patients must beware of immoderate eating and drinking, in order to avoid the plethora which, although but transient, always follows upon a free use of food or drink. How often does the long-threatening apoplexy set in in the midst of the plethora which has developed after a long and hearty meal! Here too, we must insist upon the rule of issuing the most precise orders, and of exactly regulating the quantity and quality of the meals of which the patient is to partake in future. In this connection I may mention an act of folly which I have often seen practised by tavern-keepers and itinerant wine-dealers. The latter often suppose that, by a free use of water, they can counteract the pernicious influences to which they expose themselves, although it is evident that the plethora arising after a full meal would only be increased by an immoderate addition of liquid. Besides this, however, the patient must avoid all the causes which, independently of plethora, stimulate the action of the heart, and further distend the already overcharged arteries. Under this head come the use of stimulating drinks, mental excitement, and immoderate bodily exertion. Hot water must be included in this class, and there is no wonder that the use of the Karlsbad Sprudel should make victims every year who die of apoplexy.

Besides these dietetic measures, we should see that there be no obstacle to the current of blood in the abdominal aorta, and thus remove pressure from the endangered vessels of the brain and bronchi. Besides forbidding all food that can cause flatulence, take care that the bowels be regularly moved, thus relieving the aorta and its branches from the pressure of the abdominal viscera. The practice of repeated and systematic blood-letting in treatment of hypertrophy of the heart, which originated with *Valsalva* and *Albertini*, is still adhered to by the French, but with us is falling more and more into disuse. Venesection diminishes the volume of blood for a short time only, and is apt to be followed by an erythism of the heart, and seems to favor degeneration of the substance of the organ. We do not mean that there never is any indication for bleeding, as in case of a threatened apoplexy proceeding from hypertrophy, of the heart. The application of a seton in the region of the heart is more customary with us, as soon as a hypertrophy or in fact any trouble whatever about the heart, becomes apparent. This procedure must also be pronounced useless, and even dangerous, although commanded by classical authority. Iodine and mercury are of course both inadmissible and useless. The patients often do well under the use of the "whey-cure." The grape-cure also has a good effect, but not unless we restrict the supply of other kinds of food. If we permit the patients to take their ordinary meals, and then daily to eat three or four pounds of grapes besides, dangers may easily arise, particularly that of congestion of the brain and apoplexy. I once saw a fresh attack of apoplexy occur in a person, who, after having been for four weeks at Marienbad, where he had done very well, was then daily eating four pounds of grapes at Vevay, as an "after-treatment," without, however, making any reduction in the amount of his other food.

Digitalis in pure uncomplicated hypertrophy is unsuitable. As has been brilliantly demonstrated by *Dr. Reich*, the results of experiments made with this medicine upon dogs stand in glaring contradiction to the conclusions drawn from experience at the bedside. (On the employment of digitalis in disease of the heart. Inaugural address under President Prof. Niemeyer, Tübingen, 1864.) The action of digitalis, under the use of which, in innumerable cases of disease of the heart, cyanosis, dropsy, hepatic engorgement, and suppression of urine have been made to subside, is not to lower the centrifugal pressure of the arteries, but rather to increase it. Its use is indicated in diseases in which the action of the heart is weakened, but never in cases where it is augmented. The application of cold, in the form of a tin flask filled with ice-water, and worn upon the region of the heart, is of great benefit to many patients.

## CHAPTER II.

### DILATATION OF THE HEART.

In excentric hypertrophy of the heart, the cavities of the organ become enlarged, but, as its walls at the same time are thickened by development of their muscular substance, the heart does not suffer any impairment of its functional power, which, indeed, is augmented. Although excentric hypertrophy has been called "active dilatation" by the pathological anatomists, yet, from a clinical point of view, it cannot be regarded as a dilatation of the heart. According to the general usage both of physicians and laity, the term dilatation is exclusively applied to a morbid condition of the heart, in which its cavities are enlarged, but in which there is no corresponding growth of the muscular substance of its walls, so that the contractile power of the organ is diminished. This condition corresponds to the "passive dilatation" of the pathological anatomists.

There are three recognizable forms of cardiac dilatation, although the transition of the first variety into the second is not abruptly defined:

1. The cavity is dilated, the wall of the heart retaining its normal thickness, and being merely *comparatively* too thin.

2. The cavity is dilated and the cardiac wall is *positively* thinner than is normal.

3. The cavity is dilated, while the heart-wall is *thickened;* not, however, by augmentation of its muscular substance, but through a spurious hypertrophy.

Etiology.—The causes of dilatation of the heart are—

1. When the organ is subjected to an unnaturally severe internal pressure during its diastole, causing its wall to yield to a certain degree, the fact, that contractions of the orifices of the heart, and other obstacles to the circulation, cause dilatation of the portion of the organ from which the efflux of blood is impeded, might give rise to the erroneous supposition that an abnormal resistance, encountered by the heart during its systolic movement, may result in dilatation. It is manifest, however, that, the moment that the contractile force of the heart becomes incapable of overcoming the resistance opposed by its contents, and it yields to internal pressure, the circulation of the blood will stop. The explanation of the mode in which dilatation is produced, when one of the cardiac orifices has become contracted, is as follows: The first consequence of an obstacle to the circulation is, that the affected cavity is incompletely emptied of its blood. The gush of blood which enters the heart upon diastole, instead of finding the cavity empty, finds it almost as full at the beginning of the diastolic move-

ment as it should be when that movement is complete. Blood continues to enter as long as the pressure of the afferent vessels upon their contents exceeds the power of resistance of the walls of the cardiac cavity. Let us, for instance, suppose an obstacle to exist at the root of the pulmonary artery, or, what is more likely, an obstruction in the current of the capillary system of the lungs. Such obstacle cannot prevent a systolic contraction of the right ventricle, although no doubt it may have the effect of preventing the ventricle from expelling the whole of its contents. Now, as long as the pressure upon the blood within the vena cava is greater than the resisting power of the thin walls of the right ventricle, the ventricle will be distended by an abnormal influx of blood. Moreover the diastolic relaxation is terminated by a contraction of the right auricle, whose contents are forcibly propelled into the right ventricle. As the blood enters the heart by the veins, and enters it under quite a moderate pressure, it is manifest that the right auricle and ventricle, the thickness of whose walls is only one and two lines, respectively, should be much more liable to dilatation than the left ventricle, whose ventricular wall has a thickness of five lines; and, in fact, we find that the auricles are the most frequent seat of dilatation; next to these, the right ventricle, while dilatation of the left ventricle is the rarest of all.

A considerable degree of dilatation of the left ventricle arises in cases of deficience of the aortic valves, and a smaller degree in deficience of the mitral; and this circumstance, which is taught in every text-book on pathological anatomy, also argues in favor of the correctness of the above deductions. Let us suppose that the aortic valves are insufficient, and that blood regurgitates from the aorta into the left ventricle during its period of diastole, the pressure which the blood exerts upon the relaxed cardiac wall is then a very considerable one, and capable of overcoming the resisting power of the latter. *Bamberger* has made a careful examination of fifty hearts with valvular disease of the aorta, with regard to the coexistence of dilatation and hypertrophy of the left ventricle, and has arrived at the following conclusions, which fully agree with our views as to the pathogeny of dilatation of the heart. He found that, in simple contraction at the root of the aorta, there is no dilatation, or else only a very slight dilatation of the left ventricle. Although the obstacle to the circulation is very great in these cases, there is no increase of that internal pressure, during diastole, by which alone the dilatation of the ventricle is caused. On the other hand, in insufficience of the aortic valves, there is always a considerable dilatation of the left ventricle, which predominates over the coexisting hypertrophy. There is often room enough in the dilated ventricle to contain a full-sized fist. In such cases, as we have shown, the wall of the ventricle is subjected to an extreme pressure. *Bamberger* found that the greatest degree of dilatation of the left ventricle arose when the aortic valves were the seat of simultaneous contraction and insufficience, as then such a condition would naturally be the most favorable one for the occurrence of dilatation; since the stenosis prevents a complete emptying of the ventricle during systole, while, owing to the insufficience, the blood regurgitates into the ventricle during the diastole, with all the force which the pressure of the aorta can impart.

The occurrence of a slighter degree of dilatation in insufficience of the mitral is likewise easy to account for. When the mitral valve is not effectually closed, a considerable amount of blood regurgitates from the ventricle during the systolic movement, so that the auricle and pulmonary veins become overloaded and their walls tightly stretched. Blood consequently pours into the left ventricle with unnatural force during diastole. Perhaps the hypertrophy of the left auricle and the increased energy of its contractile power also aid in causing the left ventricle to yield to the abnormal pressure, when the mitral valve is insufficient.

In stricture of the left auriculo-ventricular orifice also, there is a considerable degree of engorgement of the left auricle, and pulmonary vein, and the auricle itself becomes hypertrophied, but the augmentation of propulsive power is neutralized by the obstacle to the entrance of the blood. This is plainly the reason why the left ventricle is dilated when the mitral valve is insufficient, but does not dilate where the valve is only contracted.

Dilatation of the heart, when arising solely from an increase of the pressure of the blood within the cardiac cavities, as a rule, is soon followed by excentric hypertrophy, the continuous and abnormally active contractions of the organ giving rise to a multiplication of its muscular fibres. When we come to treat of valvular disease, we shall explain more in detail that it is by this transition from dilatation into excentric hypertrophy that the effect of obstruction to the circulation, arising from derangement of the valves, is counteracted.

2. Dilatation of the heart may arise from the loss of tone of the cardiac wall, owing to disease of its substance, in consequence of which the wall gives way even before the normal internal pressure exerted upon it during diastole. Even the simple serous infiltration to which the heart is subject in the various forms of inflammation which affect it, especially in pericarditis, diminishes the resisting power of the organ and causes it to dilate. Sometimes its muscles seem to undergo an atrophy, like that suffered by the muscles of the rest of the body after severe and protracted illness, and in consequence of which the

wall yields to the pressure of the blood; but the cause which most frequently deprives the cardiac parietes of their tenacity is degeneration of its tissues and in particular fatty degeneration.

After the subsidence of the collateral œdema which remains after the abatement of an inflammatory affection of the heart, the muscles of the organ may regain their power of resistance, and the dilatation may be repaired. In other instances hypertrophy follows upon dilatation. The dilatation arising from typhus, or protracted chlorosis, usually disappears when the attenuated muscular fibres of the heart, with the rest of the muscles, recover their proper condition. On the contrary, the dilatation proceeding from degeneration of the substance of the heart, is incapable of repair, and, indeed, always grows worse as it grows older.

3. Dilatation may proceed from the degeneration of the substance of an excentrically hypertrophied heart. This transition from hypertrophy to dilatation occurs quite as often as does the transformation above alluded to from dilatation to hypertrophy; and, indeed, it often happens that both metamorphoses take place in the same patient at different periods of the disease. Thus it can be shown that a valvular derangement first gives rise to dilatation; and that this is subsequently converted into a hypertrophy, which compensates for the deficiency of the valve; and that at last the substance of the heart undergoes degeneration, and the hypertrophy is again replaced by dilatation, whereupon the compensatory action ceases. The latter dangerous transformation often does not take place until long after the valvular disease has been established. So, too, in emphysema; years may elapse ere the excentric hypertrophy of the right ventricle, which compensates for the obstruction of the pulmonary circulation, changes into dilatation, to the great detriment of the patient. Nevertheless, it would seem that a certain period of continued overaction of the heart suffices to determine the conversion of a true hypertrophy into a spurious one, a circumstance which has not been observed to occur in other overworked muscles. Degeneration of the hypertrophied cardiac muscles is much accelerated if the patient's nutritive condition be allowed to deteriorate. One of the most common of the diseases of aged and decrepit people is an excentric hypertrophy of the left side of the heart, caused by endarteritis deformans, which, when of long standing, gradually changes into dilatation by degeneration of its muscular substance. These are the cases to which people allude when they speak of enlargement of the heart as being one of the most severe and dangerous of the maladies of that organ, and which is the most terrible bugbear of many old people.

ANATOMICAL APPEARANCES.—Care must always be taken not to mistake a heart distended by blood and relaxed from putrefaction for a dilated heart. Advanced decomposition of the rest of the body, extreme softness of the substance of the heart, and its saturation with the coloring matter of the blood, are the distinctive marks in such cases. When the dilatation involves the entire organ, its form is changed in the manner described in speaking of hypertrophy of the heart. As, however, in most cases the dilatation is partial, and far more frequently involves the right side of the heart than the left, a dilated heart usually appears wider without any corresponding increase in length. When the wall of the dilated organ seems thinned, the degree of thinning must be accurately determined by measurement, as otherwise there will be danger of error. General statements, such as "moderate thinning" or "moderate thickening" of the walls of the heart, are of no value whatever. When the wall of the left ventricle is thinned, it collapses when cut open. This does not occur when the organ is in normal condition. In cases of great dilatation of the auricles, the muscular fasciculi may be so widely separated that the walls, in places, have a membranous appearance. When the ventricles are much dilated, with wasting of the muscular substance, we sometimes find some of the trabeculæ reduced to the condition of fleshless tendinous cords.

When the wall of a dilated ventricle is thickened, it is sometimes possible to recognize that the hypertrophy is of the spurious kind, merely from the color and resistance of its substance. In other instances, the tissues of the heart appear normal upon cursory examination; but the general dropsy, and other signs of engorgement, which are not ascribable to valvular disease, or to other obstacle to the circulation, give proof of the abnormal state of the muscular substance of the heart, and microscopic examination reveals its degeneration. At other times the microscope exhibits a much slighter degree of degeneration of the substance of the cardiac wall than the intensity of the venous engorgement would lead one to suspect. As this latter condition, when unaccompanied by obstruction to the course of the circulation, is a most positive sign of lack of functional power in the heart, I feel warranted in making the following assertion, based upon a large number of accurate observations: that it is not possible, by means of the microscope, to recognize all the alterations of the muscular fibrillæ, which diminish the functional power of the heart.

Notwithstanding that the orifices of the heart also take part in the dilatation, the valves still remain capable of closing perfectly, in consequence of their enlargement, which keeps pace with their thinning, and owing to elongation of the chordæ tendineæ.

Symptoms and Course.—Dilatation of the heart renders its action more laborious, since, although its power is not diminished, the amount of blood which it must expel is increased. Hence, the effect of dilatation upon the distribution and force of the circulation is precisely the reverse of that of hypertrophy. However, as long as the substance of its walls remains healthy, the organ still continues capable of fulfilling its function, by dint of increased exertion, just as a healthy heart overcomes obstacles to the circulation by greater energy of its contraction. It is very different, however, when dilatation of the heart is accompanied by degeneration of its muscles, as it is then unable to sustain such augmentation of its functional energy. Its action is insufficient, and the consequences of this defective action become recognizable in derangement of the circulation which ensues. The amount of blood expelled from the heart being too small, the arteries are inadequately filled, their walls contract, and the size of the individual vessels is reduced. The consequence of the diminution of the arterial contents is an augmentation of that of the veins; but, as the number of the veins exceeds that of the arteries, the filling of the individual veins never increases in proportion as that of the arteries diminishes. Moreover, a part of the blood which should occupy the arteries is in the dilated and half-emptied heart. Accordingly, the signs of deficience of blood in the arterial system appear earlier and in slighter degrees of the disease than do the symptoms of engorgement of the venous system. The capillaries also become abnormally full, owing to the impediment to their circulation offered by the engorgement of the veins, while the tension of the arterial walls, even when the vessels are imperfectly filled, still exceeds that of the capillaries, so that the blood continues to flow into them. Finally, the quantity of blood set in motion by each systole being abnormally small, the circulation is retarded, and the blood acquires a more venous character, owing to the greater quantity of carbonic acid which it receives, and because it does not return so often to the lungs to obtain oxygen.

When dilatation of a part of the heart is complicated by valvular disease, emphysema, or any other affection of the lungs by which the circulation is impeded, it is often difficult to decide whether and how much the lack of blood in the arteries, the venous engorgement, the retardation of the circulation, and the venous state of the blood, depend upon the primitive disease, and how much upon the dilatation. (Thus, *Traube* ascribes the dropsy of pulmonary emphysema to dilatation and degeneration of the right heart alone, and not to destruction of a large number of the pulmonary capillaries; while, in my opinion, both of these causes are to be taken into account, and the dropsy of emphysema is only to be dreaded when disorder of the pulmonary circulation ceases to be compensated for by the hypertrophy of the right heart.) But the fact that all derangement of the circulation is averted in valvular disease or emphysema, as long as the portion of the heart involved is hypertrophied, instead of being dilated, warrants the conclusion, whenever there is much embarrassment of circulation, that either the original dilatation has not been followed by any great degree of hypertrophy, or else that the hypertrophy has turned into a dilatation through degeneration of the substance of the heart. Of course the extent of the disorders which arise in the circulatory system varies with the seat of the dilatation. In treating of valvular disease, we propose to describe more fully the conditions which result from dilatation of the various parts of the heart, just as we have made a detailed description of the effect which the state of the right ventricle exerts upon emphysema, in treating of that disease. There is but one symptom (palpitation of the heart) to which we shall draw attention at present, since its occurrence is quite as common in partial dilatation as in the complete dilatation. This distressing subjective symptom, consisting of a painful sense of pulsation in the region of the heart, often ceases when the dilated heart becomes hypertrophied, and returns when the hypertrophy begins to undergo degeneration. It is not the beat of a hypertrophied heart—which, though it often jars the chest like the blow of a hammer, is nevertheless performed without effort—which gives rise to the sense of palpitation. This sensation is rather the result of the laborious contractions of an unhypertrophied organ. Thin-blooded and chlorotic persons complain much more of palpitation than those whose heart is actually diseased; and, of all the varied disorders of the organ, dilatation, inflammation, and degeneration of the cardiac substance, are the ones which are most generally accompanied by palpitation.

In the total dilatation which generally is due to a morbid flaccidity of a degenerate heart-wall, it is often difficult to say how much of the derangement of the circulation depends upon the degeneration and how much upon the dilatation. The latter, however, plays an important part in their production, as it is found that degeneration of the heart, without dilatation (which is common enough in anæmic persons), is much better borne, and deranges the circulation much less, than when it is accompanied by dilatation.

The first symptoms which are observed in this form of dilatation consist, as we have said, of a complaint of palpitation of the heart, which form a striking contrast with the faintness of its objective and visible pulsation, and which soon is accompanied by a slight dyspnœa. The cause of the dyspnœa is easily traceable to the overloading of the pulmonary veins and capillaries, and to the retardation of the circulation. It is aggravated upon ascending stairs, walking up-hill, and similar causes, and at first is scarcely perceptible when the body is at rest. The aspect of the patient at this period is pale, owing to the lack of blood in his arteries; but the overloaded state of the veins is not at first sufficient to produce cyanosis and dropsy, although the lips may be somewhat livid. Besides this, there are a certain languor and apathy, with liability to fatigue upon slight exertion, symptoms which, as we have repeatedly said, indicate a venous condition of the blood. If the disease advance, the palpitation and dyspnœa become more distressing, the patient fears to make any exertion, because it "puts him out of breath." The lips and cheeks assume a distinctly blue tinge, the liver begins to swell, from the venous engorgement, and a slight œdema of the extremities begins to appear toward evening. In the most aggravated stage of the disease the patient complains of great shortness of breath, even when in a state of complete repose, which becomes almost intolerable upon his making the slightest effort. The pulse is small, and often is irregular and intermittent. The urine, which is extremely scanty and concentrated, deposits a copious sediment of urate of soda upon cooling, the small amount of water which it contains being insufficient to retain the salts in solution at a reduced temperature. At this period more or less albumen usually appears in the urine, and both lips and cheeks are decidedly cyanotic. The dropsy spreads from the ankles to the legs, thighs, scrotum, and abdominal integument. The upper extremities and face become œdematous, dropsical effusions also forming in the cavity of the abdomen and the serous sacs of the chest. At length the patient succumbs to the symptoms of bronchial palsy and œdema of the lungs. Every busy practitioner has repeated opportunities to witness cases where old people die of this malady with precisely such symptoms, or with symptoms but slightly different. The smallness of the pulse and the diminution of the urine are the result of the constant decrease of the arterial contents; the cyanosis, dropsy, and albuminuria are necessary consequences of the ever-increasing venous engorgement.

The symptoms of dilatation supervening upon an excentric hypertrophy, arising from endarteritis deformans, are essentially the same as those described above. It is often impossible to determine with which of these two forms we have to deal. This will not seem strange, when we consider that endarteritis deformans does not occasion any derangement of the circulation as long as the heart remains in a state of excentric hypertrophy, and that the evidence of disease only becomes apparent after the hypertrophy has become spurious by secondary degeneration, and after its compensatory action has become imperfect. When we find, by physical exploration, that an old person, suffering from cyanosis and dropsy, has a dilated heart, that his superficial arteries are tortuous, pulsate visibly, and feel hard to the touch, the case is probably one of endarteritis deformans with secondary degeneration of a heart which was once hypertrophied. If, on the other hand, there be no such condition of the peripheral arteries, the degeneration is probably the primary disease to which the dilatation is secondary.

*Physical Signs.—Inspection* never reveals the prominence of the precordial region sometimes seen in excentric hypertrophy.

When there is much enlargement, the apex of the heart is found, upon palpation, to beat abnormally low down, and too much to the outer side of the chest. Its impulse is often extremely feeble, and may even be quite imperceptible. In other cases, particularly during moments of excitement, it may be unnaturally strong (*Skoda*), and may even be equal to that of an excentrically hypertrophied heart, although, indeed, the heaving pulsation is never seen in cases of simple dilatation.

*Percussion* shows an extension of the cardiac dulness like that arising in hypertrophy, so that in general an extension of the cardiac dulness with intensification of the impulse is indicative of hypertrophy, while a similar extension with diminution of the force of the impulse signifies dilatation. Dilatation of the left ventricle, which accompanies the first stage of insufficience of the aortic valve, produces the same alterations of the percussion-sound which accompany the subsequent hypertrophy of the ventricle. The same is true of the right ventricle. In dilatation of the right auricle, the sound of percussion is dull under the sternum, and at its right edge, from the second rib to the fifth or sixth rib. Dilatation of the left auricle cannot be demonstrated by percussion, as the auricle lies too far to the rear. Upon auscultation, the normal sounds, which are loud and strong in hypertrophy, in dilatation are found to be unusually feeble, although pure; since both the auriculo-ventricular valves and the arterial walls are set into very languid vibrations by the feeble action of the heart. In other cases, the sounds are muffled; perhaps because the papillary muscles, which are atrophied as well as the wall of the heart, produce a less vigorous tension of the valves. Finally, and very frequently, too, we hear murmurs over a dilated heart instead of the normal sounds, from which, however, we are not warranted in concluding that the valves have suffered alteration in their structure. These murmurs depend rather upon the irregularity of the vibrations, into which the ill-stretched valves are thrown by the current of the blood. They are nearly allied to those which we notice in cases of abnormal innervation of the organ, where there is no dilatation of the heart, in febrile disease, or in relaxation of the cardiac muscles, such as occurs in anæmia, combined with general loss of tone of the muscular system.

TREATMENT.—The principles of treatment for dilatation of the heart are readily deducible, from the foregoing remarks upon its pathogeny and etiology. On the one hand, we have to see that the nutrition of the body goes on normally, as being the best preventive of flaccidity of the cardiac walls. On the other hand, the patient is to be protected from all causes which can render the action of the heart more laborious. Thus, a nutritious diet is very proper; but the patient must not eat much at a time, but should increase the number of his meals. Eggs, meat, and, above all, milk, are particularly commendable. Many patients do well to live exclusively upon milk for a while. Iron, which, fortunately, no longer has the reputation of being "heating," should always be prescribed when the patient shows any signs of anæmia or of hydræmia. On the other hand, all violent muscular effort should be forbidden, and the use of spirituous liquors ought to be restricted, without, however, entirely forbidding them to such patients as are habituated to their use. When the liver swells, if the feet become œdematous, or if the patient become distinctly cyanotic, digitalis should be used. Prepossessed by the doctrine of *Traube* regarding the effect of digitalis upon the contractile force of the heart, and the tension of the aortic system, I formerly regarded the use of this remedy in dilatation of the heart as unnecessary and even dangerous. Of late years I have convinced myself, from a great number of observations, that digitalis is a very efficient means of temporarily strengthening the heart's contractile power, and of thus allaying cyanosis and dropsy. In dilatation of the heart, digitalis, when combined with an exclusively milk diet, is an invaluable remedy. I have repeatedly succeeded in obtaining complete removal of dropsical effusions of great magnitude, and produced considerable temporary relief, by this mode of treatment. It were very desirable that clinical teachers and physicians should endeavor to promote the cause of therapeutics by a more rigorous analysis of the results of their treatment of disease in human beings, rather than make experiments upon dogs with their medicines. Indeed, the more recent researches of *Traube* have afforded results which are absolutely contradictory to his former conclusions, and which are more in accord with experience at the bedside.

## CHAPTER III.

### ATROPHY OF THE HEART.

ETIOLOGY.—Congenital, or original diminutiveness of the heart, which, strictly speaking, cannot be called atrophy, occurs by preference in the female sex (*Rokitansky*), accompanied by retarded development in general, but especially of the sexual organs. We know nothing of the manner in which it originates.

Acquired atrophy occurs—

1. In general marasmus, such as is developed in the course of tubercular consumption, cancerous cachexia, and cancerous suppuration, or in consequence of old age. Even attacks of acute disease, of long duration, such as protracted typhus, may cause atrophy of the heart. We also find that, although an abundant supply of nourishment is not sufficient, of itself alone, to cause the muscular tissue of the heart to grow, yet a scanty supply, or an abnormal consumption of it, may give rise to atrophy of the cardiac muscles as well, and to wasting of the muscular system at large.

2. The heart atrophies when exposed to unusual pressure from without, just as the muscles of the extremities waste away under continued compression of shackles or bandages. Atrophy of the heart also accompanies extensive pericardial effusion, fibrous thickening of the epicardium, and may even result from large accumulations of fat upon the organ.

3. Contraction of the coronary arteries causes atrophy of the heart, by limiting the supply of nutritive fluid.

ANATOMICAL APPEARANCES.—In congenital smallness of the heart, the heart of an adult may be like that of a child of five or six years of age, with thin walls, small cavities, and delicate valves (*Rokitansky*).

Acquired atrophy is almost always concentric—that is, the thinning of the wall of the organ is accompanied by contraction of its cavities. Another characteristic sign, besides the reduction in size, and by which it may be distinguished from congenital smallness, consists in the disappearance of the fat of the heart, and in the serous infiltration of the connective tissue, in which the fat formerly lay. The pericardium is opaque, the white specks, which we so often find in the heart (Sehnenflecke), are wrinkled, and the coronary arteries remarkably tortuous. The endocardium is also clouded, the valves of the veins swollen. The substance of the heart is usually pale, and its consistence less firm; in other cases, it is hard and dark. *Bamberger* very properly calls to our attention that, in many cases of concentric atrophy of the heart, there is a considerable quantity of liquid in the pericardial sac. This bears a certain analogy with the collections of water in the skull in atrophy of the brain—with hydrocephalus ex vacuo.

Simple atrophy of the heart is much more rare. Here the organ is of normal size, but its walls are thinned. Hence the normal size can only be the result of dilatation of the cavities, so that this form of disease is allied to that described in a previous chapter. This applies still more forcibly to the excentric atrophy. The latter almost completely coincides with simple dilatation. Indeed, it would be almost impossible to decide whether the walls have been thinned by excessive distention alone (dilatation), or whether atrophy of their elements have contributed to their attenuation (excentric atrophy). True, the effect of the two conditions is not quite the same, for, if the walls of a dilated heart be also thinned (as they sometimes are by accumulation of fat about the heart, and as is observed most typically in the indurated thickening of the epicardium which remains after a chronic pericarditis), we find the propulsive power of the organ to be much more reduced than is the case in simple dilatation. We have, finally, to mention that, when the contents of the left ventricle have been reduced as a result of contraction of the left auriculo-ventricular orifice, a most classical diminution in size and atrophy of the left ventricle is often observed.

SYMPTOMS AND COURSE.—According to *Laennec*, congenital atrophy of the heart is the cause of frequent attacks of fainting. According to *Hope*, besides the tendency to faintness, the signs of defective nutrition of the body, great muscular debility, palpitation of the heart, signs of anæmia, and chlorosis, are to be found in persons suffering from congenital smallness of the heart.

Acquired cardiac atrophy varies in its symptoms, according as it forms a part of a general state of marasmus, or stands alone, independent of poverty of blood or wasting of the general system. In the first instance, the symptoms are not very prominent. In fact, in certain cases, it can hardly be decided whether the enfeebled propulsive power of the heart depend upon lack of energy in its contraction, or upon atrophy of its muscles. In either case, the arteries are incompletely filled, and the blood accumulates in the veins. As, however, the blood itself is reduced in quantity, there are no signs of extreme venous engorgement. Severe dropsy, or well-marked cyanosis, is hardly ever met with in this form of atrophy of the heart. The bluish hue of the lips, the varicosities upon the cheeks of old men, the small effusions into the subcutaneous tissue in the hands and feet, which are usually cool and slightly bluish, are only partially dependent upon feeble propulsive power. Atrophy of the lungs, as we have already seen, contributes largely to the establishment of these symptoms.

Atrophy of the heart, arising in consequence of local derangements of nutrition, long-continued compression of the heart, or stricture of the coronary arteries, has a very different character. In the first place, the patient often complains of a distressing palpitation, a symptom which, as stated in a previous chapter, generally exists when the heart is unable to keep up the circulation without very great exertion.

Moreover, in consequence of the emptiness of the arteries, the veins are over-filled, and the retardation of the current of the blood gives a venous character to the latter, and occasions shortness of breath. The patients may become exquisitely cyanotic. General dropsy appears, and with it there is often great dyspnœa. If the atrophied heart be also dilated, an additional cause of engorgement of the veins and impediment of the circulation comes into play, and all the symptoms are much aggravated. But the progress of the malady is still more rapid and serious upon addition of a third cause—fatty degeneration of the cardiac muscles—the effects of which are quite similar to those of the first two, and which usually accompanies them. Such cases are tolerably common, and when old and feeble persons become blue and dropsical, without having any valvular disease, it is generally owing to dilatation and degeneration of the substance of the heart, or else to extensive endarteritis deformans. An approximate diagnosis, at least, may be made sometimes by physical exploration. As long as the patient remains at rest the heart-shock may be very feeble, or quite imperceptible. The pulse is remarkably small. In some cases, the area of cardiac dulness has decreased with the diminution of the heart, a symptom which is only of value when it can be proved that diminution of the heart has caused vicarious emphysema, distending the lungs. In other cases, instead of extension of the lung, a large effusion into the pericardium fills the vacuum caused by shrinking of the heart, and the cardiac dulness is normal. In other cases, in which the lungs are also reduced in size, the pericardial effusion may be so profuse as to render the cardiac dulness abnormally large. The same is the case in atrophy of the wall of the heart with dilatation of its cavity. As, in hypertrophy, the heart-sounds are stronger and louder, so in atrophy they are either feebler or indistinct, or else they are muffled, and sometimes murmurs are audible, which depend upon the conditions which we have already mentioned, as causes of modification of the sounds of the heart.

TREATMENT.—A real treatment of atrophy of the heart is out of the question. Of course, all violent efforts must be avoided, rich food be provided, and even the moderate employment of wine, "*vinum lac serum*," may be permitted.

## CHAPTER IV.

### ENDOCARDITIS.

ETIOLOGY.—We entirely agree with *Virchow* as to the pathogeny of endocarditis. He regards the hypothesis of the formation of a free exudation in this disease as not proven and even doubtful, and counts both this malady and the inflammation of the inner arterial tunics, from which the so-called atheroma proceeds, among the *parenchymatous inflammations*. This term is applied by *Virchow* to the active disturbances of nutrition which are provoked by an irritation, but which, instead of producing an exudation between the elements of the tissues, causes a swelling of the normal elements themselves, and a proliferation of their cells. In endocarditis the inflammation does not originate in the deeper layers of the endocardium, but upon its more superficial portions. They become enlarged, are infiltrated by a liquid whose chemical properties resemble that of mucin, that is, it coagulates into the form of threads upon addition of acetic acid. In addition to this, a vast formation of new cells takes place, which immediately organize into connective tissue. It is only in very rare cases, in the so-called ulcerative endocarditis, that the proliferation of young cells goes forward with such activity that the tissue breaks down under their pressure, producing a loss of substance, an ulceration of the endocardium.

The cause of endocarditis is somewhat obscure. It is seldom the result of direct irritation. *Bamberger* has only seen two cases of traumatic origin. The frequence with which the orifices and valves of the heart suffer from this disease scarcely leaves any doubt that endocarditis, arising from internal causes, attacks those portions of the endocardium by preference which are especially exposed to strain and friction from the action of the heart. Just as the pulmonary artery, which, though otherwise rarely atheromatous, if exposed to abnormal tension by hypertrophy of the right ventricle, is often attacked by atheroma; and just as the veins even undergo atheromatous degeneration when distended by a current of blood from a communicating artery, so in the heart, it is the narrow places, the outlets, which are most often diseased, but especially those portions of the valves which strike against one another in closing, the auricular surfaces of the mitral, and tricuspid, and the convex faces of the semilunar valves.

Whether primary idiopathic endocarditis ever occurs, and whether the disease independently can attack a previously healthy person who has been exposed perhaps to cold, may be doubted, yet it is not impossible. The great frequence of valvular disease, in individuals who profess never to have suffered from any acute sickness, makes it probable that an idiopathic chronic endocarditis is not uncommon. In the vast majority of cases endocarditis arises in the course of acute articular rheumatism (*Bamberger*), and all the more readily the greater the number of joints attacked. It is idle to indulge in speculations as to how this complication comes about, as they cannot lead to any serviceable explanation. Although, however, acute articular rheumatism is the most frequent cause of endocarditis, yet the number of cases in which rheumatism runs its course without it is far greater than was supposed for a long time, after the frequent coexistence of the two maladies had been recognized.

The fact that a blowing sound can be heard in the heart, during an attack of acute rheumatism, is not by any means a sufficient evidence of the existence of endocarditis. Such a sound, which may be heard in at least one-half of all rheumatic attacks, depends, in a very great degree, upon the irregular tension of the valves, to which the excited and uneven action of the heart gives rise. According to the careful statistical compilations of *Bamberger*, the frequence of the complication of acute articular rheumatism with endocarditis may be rated at about twenty per cent.

Next in frequence, endocarditis complicates Bright's disease, accompanying the acute form which develops after scarlatina, as well as the chronic form. Here, too, the tendency to inflammation in the heart, serous membranes, and lungs, set up by this affection of the substance of the kidney, remains inexplicable.

The endocarditis which comes on during acute febrile maladies, especially acute infectious disorders, is closely allied to the above mentioned forms. The disease seems to arise frequently from puerperal fever, while *Wunderlich* regards measles as the most prolific cause of endocarditis next to rheumatism. According to the experiments of *Billroth* and *Weber*, it seems not improbable that the blood of a fever patient acts as an inflammatory irritant, and that individuals laboring under violent fever, no matter what its cause may be, are liable to secondary inflammation of various organs, and especially to inflammation of the endocardium. If this supposition be true, the frequence of the complication of articular rheumatism and endocarditis is attributable to the intensity which rheumatic fever often acquires.

The existence of a diseased valve is frequently the cause of endocarditis. It is a matter of common experience, that a patient, at first, may have a simple valvular disease arising from acute rheumatism, but after a time, and without his having had any fresh rheumatic attack, the valvular affection become a complicated one, compelling us to infer that it is the result of a latent endocarditis.

The endocarditis which accompanies myocarditis, and pericarditis, must be considered as the result of extension of the disease by contiguity. As a very great rarity, inflammation of the lungs or pleura also spreads to the endocardium.

Anatomical Appearances.—As nearly all the congenital defects of the heart which are attributable to a former endocarditis are found in the right side of the organ, it would seem that the right side of the heart is especially liable to this disease during fœtal life. On the other hand, in extra-uterine life, it is almost as exclusively the left heart in which we find endocarditis. Inflammation hardly ever affects the whole lining of the organ, but confines itself rather to patches of varying size. As we have already remarked, however, it is from the valves, and especially from the parts of them already alluded to, that the inflammation is apt to proceed.

It has been customary to mention reddening and injection of the endocardium as the first sign of endocarditis. *Rokitansky*, however, observes, that it is only possible in rare instances to obtain a view of an endocarditis in this stage, and warns against confounding the redness of injection with the infiltration of the endocardium which takes place after death. *Foerster* points out that this reddening by injection, which we find surrounding points which have already undergone further derangement of texture, may be distinguished from the reddening of *post-mortem* imbibition, as the latter is darker and merely involves the superficial coats, while the reddening from injection exists in the deeper layers only, in which, by means of the microscope, we may see the capillaries filled with blood to bursting.

Very soon we find a puffiness and swelling of the endocardium, as its external layer thickens and enlarges. *Virchow* describes this increase in volume as "consisting of a homogeneous, translucent, tolerably clear ground-substance, in which so many cells are imbedded, that it might seem, at first sight, as though it were an accumulation of growing epithelium."

Besides this diffuse swelling of the endocardium, reddish or grayish-red delicate villi often develop as the disease advances, which give the endocardium a fine, granulated aspect, and which, sometimes, rapidly grow into tolerably thick, coarsely granular papillæ and warts. These are very hard and firm at their base, while their round, bulbous, free extremities appear soft and gelatinous. At the base we find perfectly-formed connective tissue, while the apex is still filled up by elements which have not as yet organized into connective tissue. These excrescences, known as valvular vegetations, are outgrowths from the endocardium, from proliferation of its connective tissue. Upon the auriculo-ventricular valves, these excrescences often form a border of varying width, close to the free edge of the valve, and spread hence, particularly upon the chordæ tendineæ. On the semilunar valves, they generally proceed from the noduli Morganii. We must beware of mistaking the fibrinous deposits, which are apt to form upon the rough and uneven surfaces of the valves, and almost always cover them, for the vegetations themselves.

This swelling of the endocardium, which is afterward converted from a gelatinous to a semi-cartilaginous consistence, and leads to permanent thickening and rigidity of the valves, and the retraction and shrinking of the thickened valves, in which chalky masses often form, are much the most common causes of valvular disease of the heart. When the vegetations grow old, calcification may also take place in them, so that irregular lobulated masses, of stony hardness, cover the shapeless valves. While the anatomical alterations hitherto described are the most common results of endocarditis, there may appear as less usual accompaniments of the disease—

1. Laceration of the endocardium. This may readily be accounted for, from the relaxation and softening which the endocardium undergoes. It is the chordæ tendineæ which give way with the greatest frequence; and it is easy to see that the proper tension of the valve during systole must then be materially interfered with. In other cases the valve itself tears; in others, one surface of a valve alone is torn; the blood which penetrates through the rent, causing the opposite surface of the endocardium to bulge in the form of a sac, constituting an *aneurism of the valve.* It is rare for the endocardium to give way at any point in the muscular wall of the heart—although, should this happen (but only in such a case), it may be possible for the substance of the heart-wall to take part in the inflammation—for the blood to force its way into the rupture, and more or less to tear asunder the cardiac muscles, so as to produce an *acute aneurism of the heart*, a rounded, circumscribed sac, seated upon the wall of the heart, as an appendage, bounded at its entrance by torn and ragged endocardium, its wall consisting of the forcibly separated fibres of the muscular substance of the organ.

2. The adhesions of the chordæ tendineæ, and of the edges of the valves either to one another or to the wall of the heart, to which endocarditis sometimes gives rise, are of quite as much importance, and produce consequences quite as grave, as do the lacerations; for, by adhesion of the edges of the valves, or of the chordæ tendineæ to one another, the auriculo-ventricular orifice becomes very much contracted; and, by adhesion of the valves or chordæ tendineæ with the heart-wall, closure of the mitral orifice during systole of the ventricle is rendered impracticable. We shall discuss this subject more fully while treating of valvular disease. If we reflect that the heart is in constant action, and that during the formation of these adhesions the parts must have been in a constant state of alternate contact and separation, the formation of these adhesions will appear more difficult to account for than any other anatomical change which occurs in endocarditis.

In *ulcerative endocarditis* there are irregularly-shaped, abruptly-defined losses of substance in the endocardium, which, immediately around the ulcer, is swollen and thickened. The floor of the ulcer is formed by the muscular substance of the heart, which is infiltrated with pus.

Endocarditis is accompanied by myocarditis, that is to say, the cardiac muscles take part in the inflammation with far greater frequence than was formerly supposed. At other times, the inner layers of the cardiac wall which lie next to the inflamed endocardium become the seat of infiltration, which fully explains why the cardiac wall loses its tone, and why endocarditis is apt to be followed by dilatation of the heart.

The fibrinous deposits, which almost always cover the vegetations upon the valves, may, if broken loose by the current of the blood, occasion disorder of a different kind; and baneful as the ulterior effects of endocarditis are upon the system, yet almost the only source of danger, during the height of the disease, consists in the liability of these little coagula (emboli) to wash away. Should any of them be broken off by the current and borne into the circulation, hæmorrhagic infarction and metastatic abscess will be the result. We have discussed the pathogeny of these processes in detail, while treating of metastasis into the lung. Here, however, it is not the lungs in which the infarctions arise, but, in the vast majority of cases, the embolus gets into the artery of the spleen, blocks up some one of its minuter branches, and, a wedge-shaped spot, with the apex pointing inward and the base outward, is established, which is at first of a blackish red, afterward assumes a yellow hue, and passes into a state of caseous degeneration.

We sometimes see spots of this kind in the kidney; but they are far more rare than hæmorrhagic infarction of the spleen, which is met with *post mortem*, with extraordinary frequence. In the liver they are still less common, and, as we have just observed, they are rarest of all in the lungs. In the two latter organs indeed, one could hardly conceive of the occurrence of infarction, unless a branch of the hepatic artery or bronchial artery, but not of the portal vein or pulmonary artery, were to be obstructed.

That abscesses, instead of infarction, should be so rarely found in endocarditis, is explained from what we have said already upon the pathogeny of metastasis. The embolus which here obstructs the artery does not come from a collection of putrefying material, as emboli of the lungs so often do, but consists of coagulated fibrin, a fact which is unfavorable for the conversion of the infarction into an abscess.

Should a somewhat large fibrinous clot pass into one of the carotids, or vertebral arteries, then, accordingly as the artery of the brain is totally or partially occluded, it causes the formation of hæmorrhagic foci (capillary apoplexy) with their consequences, or else gives rise to partial anæmia, and consequent necrosis of the anæmic portion of the brain (yellow softening). Indeed, occlusion of the greater vessels of the extremities, by a large embolus, may even occasion spontaneous gangrene of the toes.

We are entirely unauthorized, by the occurrence of metastasis, to infer that perforation of an exudation from the deeper layers of the endocardium to its free surface has taken place, as the coagula entirely suffice to account for the symptoms. Nor are we warranted in diagnosing a septicæmia, from the appearance in an endocarditis of signs suggestive of septic poisoning, since it is not to be supposed that any exudation which might make its way to the free surface of the endocardium could be a septic one, or could infect the blood.

Symptoms and Course.—When endocarditis supervenes upon an attack of acute inflammatory rheumatism (and, as stated above, this is by far its most common commencement), there are often no subjective symptoms to warn the patient of the new enemy which is stealing upon him, and who frequently does not declare himself in all his malignance for weeks, months, nay, for years afterward. If we ask a patient, with valvular disease, whether he has ever had articular rheumatism, he often answers in the affirmative; if, however, we ask him whether during his attack he has ever suffered from pain in the region of the heart, or from oppression or palpitation, he almost always will deny it. It is not very different if we watch the patient ourselves. Generally he does not complain, even when we make special inquiry as to the existence of this kind of trouble; and we must depend for our diagnosis upon physical examination alone.

In other instances, however, functional disturbance, more or less distinct in character, certainly does arise. Pain in the cardiac region, however, never appears to proceed from simple, uncomplicated endocarditis, even although we make pressure upon the thorax or epigastrium. In a few, but very rare instances, the frequence of the pulse increases with the commencement of the endocarditis, and may even become extremely great. We shall not lose ourselves in speculations as to the cause of this augmented frequence of the heart's action, which is sometimes enormous, nor shall we attempt to decide whether it be due to sympathy of the muscular portion of the heart, or to irritation of the ganglia seated in its walls, but shall confine ourselves to announcing the fact. It is at least equally hypothetical to assume that there is an ulcerative form of the disease in the cases of endocarditis marked by acceleration of the pulse.

As increased frequence of the heart and pulse-stroke often coexist with a reduction of the energy of the heart, which may fairly be attributed to its infiltration with serum, the pulse is frequently small, and the fever assumes the character of extreme adynamy, so that it becomes liable to be mistaken for other asthenic fevers, typhus, etc. The statement that an unobserved endocarditis is the source of many fevers spoken of as nervous, febris simplex, versatilis, torpida, putrida, etc., is an exaggeration, as the disease seldom takes the latter form. When endocarditis is attended by metastasis, especially metastasis to the spleen, the fever becomes aggravated, and rigors occur, but the presence of septicæmia cannot be inferred from such symptoms alone, since both of them arise (although they are not constant) when metastases form in the spleen, from the detachment of clots, or fragments of the valves, in cases of long-standing valvular disease, where septicæmia is out of the question.

Palpitation of the heart is a more common symptom than excitement of the pulse. The reason for this is at once clear, when we remember that the action of the heart is always embarrassed by infiltration of its muscular substance, and that palpitation is usually complained of the most when the performance of its function has become laborious, as well as excessive, and that it does not proceed from the abnormally vigorous action of the hypertrophied heart. This serous infiltration of the cardiac muscles, which sometimes arises in endocarditis, and the consequent debility and imperfect action of the heart, also account for the dyspnœa which accompanies the palpitation.

In the chapter upon hyperæmia of the lung, we have explained why these symptoms are attended by passive hyperæmia. If insufficience of the mitral actually be established while the endocarditis is still in progress, if blood be regurgitated into the auricle during the systole of the ventricle, the venous engorgement of the lungs and the dyspnœa are all the more severe.

From what we have said regarding the symptoms of endocarditis, especially from the fact that, in a great number of cases, there is absolutely no disturbance of the functions, it will readily be perceived that the malady seldom runs its course in a well-defined manner, like inflammation of other important organs. Neither does the commencement of the disease often admit of detection, nor can its progress often be followed up, nor, to say the truth, can we well fix the point where endocarditis ceases, and that malady begins which we call valvular disease. Disease of the valves is indisputably the most common sequel of endocarditis, the valves either remaining thickened, and afterward shrinking, or the chordæ tendineæ and edges of the valves adhering, or rupture of one or other of these parts occurring. As retraction of the thickened valves commences gradually, and progresses slowly, and as adhesions of the valve-tips and chordæ tendineæ only take place by degrees, it may happen that, immediately after an attack of endocarditis, there may be no perceptible defect of the valve; yet valvular disease may become apparent after the lapse of some months. If, however, the chordæ tendineæ suffer rupture, or if an orifice be blocked up by vegetations, the transition from endocarditis into valvular disease is immediate. In a previous chapter we have shown how endocarditis may cause dilatation, and afterward lead to hypertrophy of the heart.

The usual termination of endocarditis is death from disease of the valves, which is almost always its sequel; but this fatal result does not generally ensue until years have elapsed, and it is rare for a patient to die suddenly of endocarditis alone. Such a termination hardly ever takes place in the form of disease, which complicates acute articular rheumatism. It is somewhat more common in the variety which accompanies Bright's disease, or the infectious maladies, and here it usually is difficult to determine what part in the fatal issue the original disease has played, and what the complication. Palsy of the heart, engorgement of the lungs with consequent œdema, in very rare instances exhaustion through fever, symptoms of softening of the brain, of metastasis into the spleen, kidneys, and liver, even gangrene of the toes, are symptoms with which death may then take place. Recovery from endocarditis may occur often enough if the valves be spared by the inflammation. White, thickened, and opaque spots, upon the interior of the wall of the heart, are often found *post mortem*, without having produced any symptoms during life. Even inflammation of the valves may terminate in recovery, if the thickening, which probably always remains, does not derange their function. Experience does not show this termination to be common. Although the valves may act normally at first, yet they are afterward liable to become the seat of fresh irritation, until, at last, deformities arise capable of deranging their function.

We have hitherto described endocarditis such as complicates rheumatism of the joints. The functional symptoms, the progress, and the consequences of an endocarditis which complicates an existing disease of the valves present no new feature to the picture. This is also the case in that form of the malady which complicates acute infectious diseases. Here the symptoms of the main affection mask those of the complication so fully, that an exact clinical description of them can scarcely be given; in particular, the delirium, stupor, albuminuria, jaundice, etc., which certainly are very common accompaniments of this form of endocarditis, do not depend upon the endocardial disorder for their cause, but rather are a result of the infection of the blood and of the intense fever arising from such infection. Physical examination alone can give us the required information, and it should never be neglected, though no special signs demand such investigation. With regard to the origin and course of endocarditis complicating chronic Bright's disease, as this form too usually presents no subjective symptoms, it is overlooked in most cases if physical exploration be neglected.

*Physical Signs.*—The impulse of the heart in the commencement of the attack is almost always stronger and more extended than natural. The smallness and softness of the pulse, when the muscles of the heart are infiltrated with serum and contract feebly, in spite of their furious action, bear striking contrast to the above. The cardiac dulness is normal at first; but, after a few days (*Skoda*), the outflow from the pulmonary veins may be so much embarrassed that the blood accumulates in the left auricle, and the obstruction extends through the vessels of the lungs into the right heart. The right heart is imperfectly emptied, and soon becomes dilated by the blood entering from the vena cava. Hence, as we have already seen, the dulness is rendered abnormally broad. If the tissue of the valves become softened, and the valves themselves thickened by the inflammation, it is easy to see that the heart-sounds must also undergo modification. It is impossible for the softened and thickened valve to vibrate, like the hard and delicate valve. As the first sound of the heart in the left ventricle proceeds from vibration of the mitral, the substitution of an abnormal murmur at the apex for the first cardiac sound is the most frequent and important sign of endocarditis, which usually has its seat in the left heart. Besides, the thickening of the delicate web on the outer edge of the mitral prevents it from unfolding freely, and keeps the softened chordæ tendineæ from completely fixing the valve, which, if the chordæ tendineæ be broken, may even be folded backward toward the auricle during the systole of the ventricle. All these forces combine to render it impossible for the valve to perform its function during systole of the ventricle, and to prevent regurgitation of blood into the auricle. That condition, where the valve loses the power of acting as a valve, is called "*insufficience.*" If, however, the valve be but partially fixed, if part of it be free to flap in either direction, if some of the blood pressing against it be opposed by a portion only of its lower surface, while the rest, flowing back into the auricle, bathes its upper face, the vibrations of the mitral become entirely abnormal and irregular, and give rise to another murmur, which takes the place of the first sound of the left ventricle. We have seen that the second sound that we hear at the apex is, under normal conditions, produced by vibration of the semilunar valves of the aorta, whence it is conducted to the apex. In the normal heart, the entrance of blood into the ventricle is unaccompanied by any murmur or other sound. If, however, in endocarditis, the auricular face of the mitral-valve be studded with warty excrescences, and if the blood have to flow over a rugged surface instead of a smooth one, friction of the blood-stream produces a murmur which is audible at the apex during the diastole of the ventricle. The second tone, propagated from the aorta, may also be heard with it, or the latter may be drowned by the intensity of the new murmur, and thus be imperceptible. The larger the excrescences, and the more they encroach upon the orifice, so much the more intense is the friction of the blood, and so much the louder the murmur. In the extremely rare cases in which the right ventricle is the seat of endocarditis, similar symptoms may be made out at the lower part of the sternum, where we listen to the sounds of the tricuspid. It would be exceedingly difficult, however, to make a diagnosis here, as the right ventricle is hardly ever the sole seat of disease, and we should scarcely be able to distinguish whether the sounds were conducted from elsewhere or actually originated at the tricuspid. The sounds of the aorta are usually pure, as its valves are far more seldom attacked by endocarditis. Should it occur, however, should warty growths form upon the lower sides of the semilunar valves, a murmur, produced by friction of the blood upon these asperities, arises during systole of the ventricle, which is best heard at the root of the aorta—i. e., at the sternum, on a level with the second intercostal space, and which is conducted hence along the carotids. It is much less common to hear a diastolic murmur at this point than a systolic one.

We hear normal heart-sounds at the pulmonary artery almost always, as the disease hardly ever extends as far as this. On the other hand, we often hear a remarkably loud and sharp accentuation of the second sound of the pulmonary artery, which is a sign of importance. The fuller the pulmonary artery becomes, so much the stronger does the shock grow which its semilunar valves must sustain during diastole. Now, as an acute insufficience of the mitral develops in the majority of cases of endocarditis, the pulmonary artery must suffer distention and its second sound must become intensified.

Diagnosis.—Endocarditis occurring in the course of acute rheumatism is often overlooked, and quite as often its presence is diagnosticated where it does not exist. In order to avoid the former error, never fail to auscult all patients with acute articular rheumatism daily, even in the absence of all complaint or constitutional disturbance. That you may not rush from Scylla into Charybdis, however, beware how you declare an endocarditis upon the mere occurrence of a blowing sound, audible at the apex. The symptom may, indeed, be due to thickening of the valve by inflammation, but it is quite as likely to be dependent upon mere abnormal tension of a healthy valve, caused by violence of fever or irregular action of the heart. Neither condition can be determined from the quality of the murmur, and diagnosis remains a matter of doubt until the signs of dilatation of the right ventricle and overloading of the pulmonary artery, lateral extension of cardiac dulness and intensification of the second pulmonary sound, supervene upon the murmur.

The differential diagnosis is still more difficult between a recent endocarditis complicating articular rheumatism and an old valvular derangement, which happens to preëxist, especially insufficience of the mitral. Such cases are by no means rare. There are few maladies which have so great a tendency to relapse as acute articular rheumatism; indeed, we meet with sufferers who have had attacks of it, of more or less severity, every year since its first onslaught. If we have not previously seen or examined them, and if upon some fresh relapse we hear a systolic blowing at the apex, the cardiac dulness extending laterally, and the second pulmonary tone being sharply-accented, we must remain in doubt, unless the signs of dilatation of the right ventricle have attained such a height as cannot be ascribed to acute insufficience. In other cases we may perhaps ascertain if, after any of his previous illnesses, the patient have remained short of breath, etc.

PROGNOSIS.—Rarely as life is threatened by endocarditis itself, the prognosis of this malady as to complete recovery is bad. Indeed, in the cases in which the disease is recognized, it almost always leaves derangements behind it, which sooner or later imperil life. Endocarditis, which attacks the wall of the heart, is, no doubt, far less dangerous; but it occurs rarely, and, moreover, is quite unrecognizable.

Symptoms which would lead us to fear an unfavorable termination to this disease are those which indicate considerable implication of the muscle of the heart in the inflammation, such as an extremely frequent pulse with scanty filling of the arteries. Rigors are quite as ominous, indeed more so, as well as acute swelling of the spleen, or pain in that region, vomiting, or the appearance of blood or albumen in the urine, or symptoms of hemiplegia, in short, the signs of metastasis.

TREATMENT.—The indication as to cause in treatment of endocarditis cannot, as a rule, be met. A genetic connection undoubtedly exists between acute articular rheumatism and this disease, whether the former merely predispose to the latter, or whether the alliance be still more intimate. Great, however, as is the number of remedies and modes of cure recommended for rheumatism, it is only equalled by their untrustworthiness. We are no less helpless against the morbus

Brightii, the acute exanthemata, and the other infectious maladies, which give rise to endocarditis, or, at least, predispose toward it. With regard to the indications from the disease and the antiphlogistic apparatus, we have already and repeatedly declared that the majority of the so-called "antiphlogistics," and, above all, venesection, often as they are employed in inflammation, have no right to the name. But, in spite of contrary assertions on the part of French and English physicians, there is, perhaps, no affection in which the practice of bleeding without special occasion, as well as the employment of calomel and "blue ointment to reduce the plasticity of the blood," is so dangerous as in endocarditis; and we must entirely agree with *Bamberger*, when he states his belief that most patients, who die during an attack of this malady, have perished less from the disease than from the treatment.

Even local blood-letting should only be resorted to where there is pain about the heart, and here we generally have to do with complications. With regard to cold, which we have employed against inflammation of internal organs as freely as it has been used in inflammation of external parts, we do not apply it in these cases, unless especially demanded by extreme excitement of the heart's action, inasmuch as, according to our experience, even when applied upon inflamed joints in rheumatism, it has but trifling palliative effect. Indeed, although many cases of endocarditis, which used formerly to escape diagnosis, are now recognized through pleximeter and stethoscope, yet their treatment is no more successful than before; nay, if the physician find the evidence of the presence of endocarditis an occasion for meddlesome treatment, it were better for the patient had the doctor never learned auscultation.

The indication as to symptoms calls for venesection in cases wherein overcharge of the pulmonary circulation imperils life by threatening œdema of the lungs, and demands prompt relief by diminution of the volume of the blood. A great acceleration of the pulse and signs of feebleness in the action of the heart, cyanosis, etc., indicate the exhibition of digitalis. Should palsy of the heart threaten, stimulants must be used.

## CHAPTER V.

### MYOCARDITIS.

Etiology.—Myocarditis consists of an inflammation of the muscular fibres of the heart, whereby they are softened, become flabby, and finally disintegrate. This destructive process is accompanied by proliferation of the perimysium; the gaps formed by absorption of the primitive fasciculi are filled up by connective tissue, and thus a scar is formed in the heart-wall; or else the perimysium breaks down simultaneously with the muscular fibrillæ, and a mass of *débris* collects in the substance of the wall. This is called an abscess of the heart.

Myocarditis is not a rare affection, and we find *post-mortem* signs of its former existence in many cases of valvular disease of the heart resulting from endocarditis. Indeed, the etiology of myocarditis is, in a great measure, identical with that of endocarditis, acute articular rheumatism acting most frequently as the cause in either disease. Myocarditis, thus excited, usually appears in the form of mere circumscribed spots, which terminate in scar-like alterations of a portion of the cardiac wall; but in more rare instances it may result either in extensive degeneration, which may give rise to a chronic aneurism of the heart, or else may produce cardiac abscess. In most cases we may regard myocarditis accompanying acute rheumatism as an extension of a concomitant endo- or pericarditis. In other cases, however, the disease runs a more independent course, is more extensive than any attendant endo- or pericarditis, which, in their turn, may then be considered as depending upon the inflammation of the heart's substance.

Chronic disease of the heart, particularly valvular disease, leads to myocarditis, and to formation of scars in the heart quite as often as to endocarditis.

Emboli, proceeding from gangrenous lungs, not unfrequently enter the coronary arteries of the heart, and we then see numerous abscesses in its wall, as well as abscesses in many other organs of the aortic circulation.

Septicæmia, protracted typhus, tedious and malignant scarlatina, even though the occurrence of embolism be not proved, nay, though it be very unlikely, may also give rise to abscess of the heart. The pathogeny of such abscesses is obscure.

In the second volume we shall treat of syphilitic myocarditis, when we come to treat of syphilis in general. Traumatic myocarditis, like traumatic endocarditis, is one of the greatest of rarities.

Anatomical Appearances.—The seat of myocarditis is almost exclusively the left ventricle, especially the apex; but quite as frequently (according to *Dittrich*) it occurs in the septum just below the aorta. The papillary muscles, however, are often affected by the disease, which fact is of importance to the pathogeny of deformity of the valves.

At the outset of the malady, the muscular substance appears of a dark bluish-red hue. Soon, however, the injection disappears, and discoloration of the muscular fibre arises, the diseased place becoming of a grayish color and softened. Under the microscope, after the transverse and longitudinal striæ have disappeared, we see the fibrillæ broken down into a finely-granular detritus, with a few fat-globules. We can rarely get opportunity to observe myocarditis in this stage. Much more commonly we find its results, in the form of irregular, ramifying collections, varying in size, of a reddish-white or white color, and of a scar-like density, scattered through the muscular substance of the heart. Sometimes this indurated tissue is spread over a large portion of the heart-wall, and forms its sole component. Here the degenerated wall may yield to the pressure of the blood; a protrusion may form, and a *true aneurism* of the heart result, which is to be distinguished as *chronic cardiac aneurism* from that form described as *acute cardiac aneurism*, in treating of endocarditis. Such sacs may attain the size of a hazel-nut or even that of a hen's egg, or larger. The scar-like walls usually grow thin from distention; they sometimes ossify, and quite often their cavity contains masses of stratified fibrin, such as we find in aneurism of arteries. The entire heart is generally dilated as well as the aneurism, and, even when there is no aneurismal pouch, numerous scars in the heart-wall will cause dilatation of the organ. On the other hand, large scars in particular situations, as at the approach to the aorta, may cause diminution of the capacity of the heart (*Dittrich's* true cardiac stricture).

When endocarditis terminates in abscess, discoloration and softening prevails more and more in the muscle, until at last a collection of yellow, purulent liquid, surrounded by softened and discolored muscular substance, is formed. Such an abscess rarely becomes incapsulated and dries up; perforation nearly always takes place, unless death occur beforehand. If the perforation be into the pericardium, pericarditis follows; if into the cavity of the heart, the *débris* of its broken-down tissue passes into the circulation, and numerous metastases are often the consequence. The insertion of an aortic valve may be torn away by the inward pointing of an abscess; or communication between the two sides of the heart may be set up; even the entire cardiac wall may suffer rupture. Tearing up of the muscular structure of the heart by infiltration of the blood, which we have described in a previous chapter as acute cardiac aneurism, may, of course, occur with equal or even greater ease in consequence of the pointing of such an abscess to the interior.

SYMPTOMS AND COURSE.—Myocarditis is but seldom diagnosticated with certainty during life. As a mild form of the disease complicates almost every case of endocarditis, we seem warranted in the inference that the substance of the heart is more seriously inflamed when the region around it appears unduly sensitive (which it never is in pure endocarditis), still more so if there be great acceleration of the pulse, if the pulse grow small, or, above all, if the heart's action become irregular. Even then, however, our opinion will only amount to a somewhat vague suspicion. Diagnosis of myocarditis grows more sure, but not certain, when symptoms appear in the course of acute rheumatism which suggest diseases of the heart, while physical examination affords negative evidence of endo- or pericarditis. If, now, rigors should set in, or swelling of the spleen, or pain in the region of the spleen, vomiting, or pain in the region of the kidneys, with the presence of albumen and blood in the urine; in short, if metastases be established, the diagnosis becomes tolerably certain: but such cases are not common.

If cicatrices have formed at numerous points in the heart, and if the heart be dilated in consequence, symptoms of dilatation appear, such as we have already described, only they are more severe; and it is impossible to say, in most cases, what parts the dilatation and degeneration respectively play in retarding the circulation and overloading the venous system. Thus, in the diagnosis of mitral insufficience, we may bear in mind that it may possibly have been induced by degeneration of the papillary muscles. Extensive scar-like degeneration of the heart-wall, as well as true cardiac stenosis of *Dittrich* and chronic cardiac aneurism, causes symptoms of extremely depressed action of the heart. The beat is scarcely perceptible, the arterial pulse is extremely small and weak as well as very irregular and intermittent. Extreme cyanosis and general dropsy accompany these symptoms. If called upon for a diagnosis in a case of this kind, after exclusion of valvular deformity as a cause of the derangement of circulation, we must count diffuse cicatricial formation as one of the alterations of structure capable of producing the train of symptoms above described; but we shall hardly ever make an absolutely certain diagnosis by the system of exclusion of other anatomical changes, such as dilatation with atrophy, extensive fatty degeneration, etc., etc.

As for abscess of the heart and the various results of perforation, we are rarely able to form more than a vague diagnosis after the numerous metastases have arisen. We have no means of ascertaining this condition with certainty.

TREATMENT.—We can hardly speak of the treatment of myocarditis, having almost denied the possibility of recognizing its existence. Should it be possible to diagnosticate the disease, the treatment would not differ from that of endocarditis. It is, of course, out of our power to remove the cicatrices, or to avert the embolism from perforation of an abscess, or to allay its effects. A mere treatment of symptoms is all that can be effected.

## *VALVULAR DISEASE OF THE HEART.*

By valvular disease of the heart, in its narrowest sense, we mean merely those anomalies of its valves which affect the function of the organ, and thus react upon the circulation. Valvular anomalies which give rise to no symptoms, and which hence are purely matters of pathologico-anatomical curiosity, and not of clinical interest, need but little notice in the following chapter. They are—

The so-called simple hypertrophies of the valves, which are found chiefly upon the mitral near its free border, whence is found growing a series of little lumps of a jelly-like connective tissue. The fine web on the lower border, upon whose unfolding the valvular action mainly depends, remains intact in hypertrophy, while endocarditis usually has the effect of thickening it, and, as it were, rolling it up.

The next deformity of the valves, which does not derange their action, is enlargement, which often occurs in them with simultaneous thinning, when the ostium is abnormally dilated. Most cases of perforation of the valves also belong under this head. Small oval fissures or holes are often seen in them, which, however, do not seem to impair their efficiency.

The most important valvular changes are those known as *insufficience* and *contraction*. These two alterations nearly always coexist, one usually prevailing over the other, however, in degree. By insufficience, we mean that condition of a valve which renders it incapable of preventing regurgitation of blood into the cavity which, as a valve, it should close. If the entire contents of the ventricles be not thrown into the aorta and pulmonary artery during systole, and if a portion of the blood regurgitate into the auricles, the mitral or tricuspid are insufficient. Again, if, during diastole of the ventricle, part of the blood which had entered the aorta and pulmonary artery flow back into the ventricle, the semilunar valves are insufficient.

By stenosis (constriction) of a valve, or, more properly speaking, of an orifice, we mean that condition by which the effluent blood meets with abnormal resistance through contraction of the outlet of the heart.

Although valvular deformities have, in common, the effect of retarding the circulation, the influence upon the distribution of the blood varies according to the seat of the affection. The system can endure valvular deficience at one point much better than at another; hence we deem it better at once specially to describe its effects at the different outlets, rather than to go into a further general discussion of the subject. We cannot altogether avoid repetition, by this method of treating the subject, but shall thus have less to repeat, and, moreover, less to retract, than by any other mode. As the pathogeny of valvular defect of the aorta is much more simple than that of mitral disorder, as its symptoms are easier of comprehension, and as its consequences are much longer and better withstood than are those of mitral deficience, we shall first take up the subject of aortic valvular disease. Derangement of the valves is of far less common occurrence in the right than in the left heart, so that we shall reserve the discussion of the former until the last.

## CHAPTER VI.

### INSUFFICIENCE OF THE SEMILUNAR VALVES, AND CONSTRICTION OF THE AORTIC ORIFICE.

ETIOLOGY.—Closure of the semilunar valves takes place in a manner purely mechanical, while a certain vital action is required to effect closure of the valves between ventricle and auricle, namely, contraction of the papillary muscles. If the mere pressure of the blood during diastole of the left ventricle do not suffice to deploy and press together the leaves of the semilunar valve, which were pushed up against the wall of the aorta during systole, there will be regurgitation of blood into the ventricle, and the valve is insufficient. If, however, during systole, the semilunar valves do not yield to the current of the blood, and lie back against the aortic wall as it emerges from the left ventricle, but stand projecting into its outlet, we have constriction (stenosis). Much more rarely the latter occurs from contraction of the aorta at the point of insertion of the valves, whereby the outlet is diminished.

The alterations which cause insufficience and constriction of the aortic valves are the results of inflammation, but less often of endocarditis, which we have described in Chapter IV., than of a more chronic form of inflammation, which attacks the arteries, and whose results are known as *atheroma of the arteries.* Hence it follows, although not without exception, that valvular disease of the aorta is found at a more advanced period of life, when arterial atheroma is far more frequent than during youth, and that its development is more slow and gradual than that of the disorders caused by endocarditis.

ANATOMICAL APPEARANCES.—If, upon autopsy, we remove the heart and aorta; and if, upon filling the latter with water sufficient to distend its walls, the water flow into the ventricle because the edges of the valves do not touch, we may assume that such regurgitation has also occurred during life, and must regard the valves as insufficient.

The anatomical changes which cause insufficience are usually shrinking and shortening of the valves, so that, even if spread out by the blood, they would not meet. But thickening, and rigidity too, may prevent closure, the pressure of the blood becoming insufficient to make the leaves flap together. Much more rarely we find adhesion of the valve to the arterial wall, or laceration or detachment of one of the leaves from its insertion, as a palpable cause of insufficience.

Besides these changes at the root of the aorta, we constantly find in the cadaver a degree of excentric hypertrophy of the left ventricle greater than is observed under almost any other circumstances. The wall of the ventricle may be an inch in thickness, its cavity is often capable of containing a fist. We have already seen that dilatation of the left ventricle is the necessary result of severe pressure induced by it from within while in a state of relaxation, and that hypertrophy follows in consequence of the augmented effort which it must make in order to propel the increased volume of blood which it holds. A large number of the signs of aortic insufficience are due to this enormous hypertrophy of the left ventricle. In a former chapter we have fully detailed all the alteration which the shape of the heart undergoes from this enlargement. We have seen that the rest of the organ participates in a less degree in the affection, and that bulging of the septum into the right ventricle materially encroaches upon the capacity of that chamber.

The mouth of the aorta may contract to such a degree as barely to admit the insertion of the end of the little finger into the narrowed opening. The anatomical changes which occasion such strictures are generally the thickening and shrinking of the flaps described above. These flaps may form unyielding prominences at the root of the aorta; so that it becomes equally impossible for the stream of blood to lay them back against the aortic wall during systole, and for the weight of the blood during diastole to force them together again. Cohesion of the semilunar flaps is the next cause of stenosis, and is the more marked the more the point of adhesion approaches the centre of the valve. Old vegetations on the valves, of cartilaginous hardness, and which are often the seat of calcareous deposit, assist in blocking up the constricted passage, although they rarely constitute the sole cause.

In simple stricture of the aortic valve, the left ventricle has no increase of pressure to support during diastole, and hence does not become dilated; it has, however, to propel its blood through a contracted orifice, and becomes hypertrophied on account of the greater amount of effort thus required from it. In contradistinction, then, to what we meet with in insufficience of the semilunar valves, we find a simple hypertrophy, instead of excentric hypertrophy of the left ventricle, when the aortic outlet is contracted.

We have said above, that the two forms of valvular derangement usually coexist; as, however, insufficience soon predominates over stenosis, we find a gradual transition from simple to the most intense excentric hypertrophy taking place in the left ventricle.

SYMPTOMS AND COURSE.—The ultimate effect both of stenosis and of insufficience of the aortic valves must always be a retardation of the circulation; the blood returns to the lung with diminished frequence, and hence assumes a more venous character. (Of course, with every systole, an abnormally small amount of blood is discharged from the ventricle, or a portion of it flows back again during diastole.) The consequences are, that the aorta and its branches are inadequately filled, while, on the other hand, the pulmonary vein is gorged with blood, which is prevented from flowing away into the left auricle, already almost full. Thus the entire pulmonary system becomes overloaded; but, being incapable of containing the whole of the blood which should properly fill the aorta, the remainder gradually accumulates in the veins of the aortic system, and gives rise to cyanosis, dropsy, etc.

As a rule, however, nothing of this kind takes place, until after the lapse of considerable time; inasmuch as simultaneous hypertrophy of the left ventricle has the opposite effect, and neutralizes the baneful influence of the defective valves. While the latter tends to retard the circulation of the blood, and to render it venous, hypertrophy accelerates its course and makes it arterial. While valvular deformity causes decrease of the contents of the aorta, hypertrophy renders the aorta fuller; while deficience of the valves hinders the outflow from the pulmonary veins, and lets the lesser circulation overcharge itself with blood, hypertrophy facilitates such outflow, and relieves the pressure upon the pulmonary system.

By keeping these facts in view, it is easy to understand how it happens that persons with extreme deficience of the valves of the aorta enjoy comparatively good health, if only there be a compensatory hypertrophy of the left ventricle; and, indeed, such persons are frequently not even short of breath, a symptom never missed in cases of valvular disease of the mitral.*

There may be some palpitation of the heart, but it is not constant. It is very remarkable, too, that the patients complain so little of jarring of the thorax. Sometimes attacks of pain in the chest and left arm occur, which we shall describe more closely in the chapter upon angina pectoris.

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* A huntsman in Greifswald, who suffered from extensive stenosis and insufficience, and immense excentric hypertrophy of the left ventricle, performed all the manœuvres and forced marches of the army without difficulty.

This state of comparative good health is common to both stenosis and insufficience; in general, however, the symptoms differ widely, those of one or other malady usually predominating. Insufficience gives rise to symptoms, and dangers which proceed from the consecutive excentric hypertrophy, which, no longer merely compensating the disorder of the valves, produces an excessive action of the heart. The patients then usually complain of dizziness, headache, and of spots before the eyes. In other cases, they suddenly perish from apoplexy. More rarely asthmatic attacks occur, but all these symptoms are due to the hypertrophy (Chap. I.), and not to the valvular disorder.

In stenosis, on the other hand, the symptoms of the circulatory impediment outweigh those coming from the hypertrophy, and although a patient may do well for a considerable length of time, evincing no signs of venous engorgement, yet there will be tokens that the arteries are but scantily filled, a symptom which must always precede those which indicate overcharge of the veins. The patients look pale, are prone to fainting-fits, and present signs of anæmia of the brain; just as others, who suffer from insufficience of the valves, seem to incline to cerebral hyperæmia and to apoplexy.

This period of comparative comfort, enjoyed by patients with disease of the aortic valves, often ceases in a somewhat sudden and remarkable manner, after having lasted, perhaps, for many years. Either because the hypertrophied heart has degenerated, or else from insufficience of the mitral, caused by chronic endocarditis, which so often complicates valvular disease, or through increase of the original aortic defect, or finally because extensive atheroma of the aorta has set in, thus giving rise to a new hinderance to the circulation, the hypertrophy of the left ventricle is at last no longer able to compensate for the deficience of the valves, and to overcome the impediments to the circulation. Then the symptoms appear which we have mentioned at the beginning of this article. The patients grow short of breath, the veins of the aortic system become overloaded, cyanosis and dropsy arise. These symptoms set in much sooner in mitral disease, and hence shall be described in the next chapter.

Death takes place either from œdema of the lungs (or else, when there is insufficience, by apoplexy). Frequently, too, death results from embolism, to which valvular disease of the aorta gives rise with a frequence next to that of endo- and myocarditis. In most of the cases wherein embolism of the arteria fossæ Sylvii has been found to have caused necrosis of the brain, valvular disease of the aorta has existed.

*Physical signs of insufficience of the aortic valves.—Inspection* and palpation furnish the usual signs of hypertrophy of the left ventricle, namely, prominence of the cardiac region, an impulse often enormously increased, and which shakes a broad tract of the thoracic wall and sometimes actually lifts it; considerable descent of the apex, even as far as the eighth rib, with displacement outward. *Percussion* also shows an elongation of the heart, where the lower limit of dulness is not obscured by the position of the left lobe of the liver. Upon *auscultation* (best at the right edge of the sternum, at the second intercostal space), instead of the second sound, we hear a murmur, arising from irregular vibrations caused by imperfect tension of the roughened, misshapen valves. In very rare instances, besides the murmur, we hear the normal second sound of the heart, although but feebly, and this occurs, as it would seem, when one or other of the valves continues sound, and is thrown by the blood into its normal state of vibration. The murmur is usually conducted both to the apex and along the sternum, and may even be heard at the sides of the chest and along the back-bone. The first sound, as heard at the aorta, is pure in the few cases in which insufficience exists without constriction of the valve or roughness upon its under surface. In the majority of cases, however, it has undergone the modifications peculiar to constriction of the aortic orifice. The first sound of the mitral is inaudible in many cases, a fact accounted for by the following excellent explanation of *Traube:* As the left ventricle is supplied from two sources during diastole, as it receives blood both from the auricle and from the aorta, the force of its internal pressure soon exceeds that with which the blood enters the ventricle from the auricle. A reversed current is thus established, flowing from ventricle to auricle, and which shuts the mitral valve before the diastolic movement is complete. Sometimes, besides the diastolic murmur, another sound is heard, caused by the premature closure of the mitral valve. Unless there be some complication, the sounds of the pulmonary artery are normal. The phenomena observed in the peripheral arteries, although chiefly dependent upon the consecutive hypertrophy of the left ventricle, are very characteristic in insufficience of the aortic valves. The carotids often pulsate in a remarkable manner. If we listen, we do not hear two distinct tones, as we should do under normal conditions (one supposed to proceed from the vibrations of the wall of the carotid, expanded by the blood-wave; the second, attributable to conduction of the second sound of the semilunar valves). The second sound is not heard, as the semilunar valves do not vibrate normally, or, as more rarely happens, we hear a murmur which takes its place. According to *Bamberger*, the first sound is also deadened in the carotids or turned into a murmur, a phenomenon which he attributes to immoderate tension of the carotid walls. Even the smaller arteries at a distance from the heart produce a sound, during their expansion, by the vibration of their walls. Their tortuous course and their pulsation, visible at the radial artery, and even in smaller arteries, are also strikingly characteristic symptoms of aortic insufficience. All these phenomena, excepting the diastolic murmur conducted to the carotids, occur also in hypertrophy of the left side of the heart, when there is no insufficience of the aortic valves; but there is one symptom appearing in the arteries which is pathognomic of the valvular disorder in question. This consists in a remarkably rapid subsidence of the arterial expansion, which, indeed, is of but momentary duration. This jerking pulse (*pulsus celerrimus*) depends upon the fact that the artery, distended during systole of the ventricle, is emptied in two directions during diastole. In some cases of insufficience of the aortic valves the physical signs of excentric hypertrophy of the left heart are less distinctly marked. The apex beats in the fifth or sixth intercostal space, the impulse is not of a heaving character. Such patients usually suffer from dyspnœa because the valvular disease is not compensated for, and the lungs are loaded with blood. We are unable to account for this exception to the rule, which is not uncommon.

*Physical signs of stricture of the aortic valves.*—*Inspection* and *palpation* show signs of simple hypertrophy of the left heart. The impulse is stronger, the apex dislocated downward and outward, but not as much so as in insufficience. Upon palpation we often feel a distinct whizzing about the aorta accompanying systole, which is rare in insufficience. Upon auscultation we hear a systolic murmur over the valves of the aorta, which is usually very loud, and extends so as to be heard all over the region of the heart, masking the other signs. During diastole of the ventricle, as the stricture is seldom uncomplicated, sometimes we hear a feeble sound, but far oftener a murmur. In the carotids, the systolic murmur is sometimes, but not always, conducted to the ear from the aorta; or we sometimes hear a short, ringing sound in its place. The second sound, too, is usually inaudible in the carotid. The pulse is as small and incompressible as it is hard and full in insufficience.

TREATMENT.—Treatment of insufficience of the aortic valves is essentially like treatment of cardiac hypertrophy. Immoderate eating and drinking, and bodily and mental excitement, are to be avoided with care; determination to the head is to be averted, by daily evacuations of the bowels; venesection is never to be practised, unless the brain be endangered by immoderate "rush of blood." In this respect we should be the more cautious, as it is almost certain that the practice of bleeding favors degeneration of the heart; and attenuation of the blood undoubtedly promotes the tendency to dropsy.

Stricture of the aortic outlet requires measures of quite a different nature. Here we have no threatening hyperæmia to allay, or over-action of the heart to moderate. Much more depends upon furthering the nutritive state of the system, and, with it, that of the heart, so that its contractions may have force enough to prevail over the resistance at the outlet. Rich animal food, and even the moderate use of wine, are quite as strongly indicated here as they are contraindicated in insufficience. Blood-letting must never be practised. Use of digitalis is to be confined to those cases in which compensation begins to become imperfect. It is most effective in the cases in which the action of the heart is so accelerated that the left ventricle apparently has not the time to expel its contents through the narrowed opening during the short period of systole.

## CHAPTER VII.

### INSUFFICIENCE OF THE MITRAL VALVE, AND CONSTRICTION OF THE LEFT AURICULO-VENTRICULAR ORIFICE.

**Etiology.**—The mode of origin of insufficience of the mitral is, in many cases, quite analogous to that of insufficience of the aortic valve; in other cases, however, it depends upon a morbid state of the papillary muscles and chordæ tendineæ; and, indeed, there have been instances in which, although during life the valve was deficient, yet after death no palpable alteration in it could be detected. Stenosis of the auriculo-ventricular passage, which is often found to accompany insufficience, arises partially through contraction of the ring of valvular insertion, partially through adhesions of the valve-tips, or chordæ tendineæ.

Valvular disorder of the mitral is almost always a consequence of endocarditis, or of myocarditis; more rarely of atheromatous degeneration. It is only when valvular disease of the aorta accompanies similar disease of the mitral that the latter depends upon the chronic form of inflammation caused by atheroma.

**Anatomical Appearances.**—The most common lesion found in mitral insufficience is a marked shortening of the valve-tips, the valve itself being thickened and indurated, often enclosing large, flat plates of calcareous matter. The delicate, tender web on the free edge of the valve has disappeared, the edge forming a thick, clumsy pad, upon which the chordæ tendineæ originating from the papillary muscles are inserted. Of the secondary chordæ tendineæ, which, springing from the primary set, are inserted into the web of the valve, there is hardly any trace. In other cases, instead of these lesions, or, in addition to them, the valve is torn. Still oftener, it is the chordæ tendineæ that have given way, and it can be distinctly recognized that the latter, which are usually thickly covered by the vegetations previously described, are inverted by the regurgitating stream of blood, and made to flap backward into the auricle. More rarely, the tendons are adherent to the wall of the heart, so as to prevent the valve-tips from approaching one another. Finally, as more or less extensive tendinous degeneration of the papillary muscles not unfrequently constitutes a minor source of the disorder, and where neither these nor other anatomical alterations are found to account for an insufficience which has notoriously existed, it is most probable that some invisible change in these muscles has been the cause of the symptoms. The lesions, which the cavities and walls of the heart exhibit in cases of insufficience, are equally characteristic and interesting. The left auricle, into which the blood is first driven during systole, is always a good deal enlarged, and its walls are considerably thickened. The pulmonary artery and vein are in like manner dilated, as is also the right heart, both ventricle and auricle. The right ventricle, whose task is enormously increased, becomes so much hypertrophied that its walls grow as thick as those of the left. If cut open, they do not collapse as before, but the cut gapes as it would do if made in the left ventricle. There is almost always a moderate degree of dilatation of the left ventricle, into which, as we have seen, the blood pours under greatly-increased pressure.

In insufficience of the mitral valve, the valve-tips are shortened; in constriction of the orifice, they have generally grown narrower, and this contraction of the valvular ring is the most common cause of impediment to the flow of the blood from auricle to ventricle. It rarely happens, however that the valves thus thickened by endocarditis, and in which new connective tissue is growing, contract in one direction alone; they almost always become narrowed and shorter simultaneously, so that stenosis and insufficience appear together. In other cases, the lower edges of the valve-tips, or of the chordæ tendineæ, are so intimately united, that the valve takes the shape of a funnel, broad toward the auricle, and ending toward the ventricle in a narrow opening, through which it is often almost impossible to pass the tip of the finger. The vegetations, which often cover the valve in the form of hard, wart-like concretions, may also contribute to occlusion of the orifice.

Dilatation of the left auricle, and of the pulmonary arteries and veins, is also a constant accompaniment of stenosis of the mitral, and the walls of the dilated chambers exhibit hypertrophy similar to what we have described above. The left ventricle, however, is in a condition opposite to that which we find in insufficience. Instead of being hypertrophied and dilated, it is generally small, and its walls are thinner, rather than thicker. We have already accounted for this circumstance. In spite of the violent pressure under which the blood is thrown into the ventricle, its walls encounter a moderate pressure only from within, as the increased propulsive power is neutralized by the greater resistance met with in the contracted ostium.

Symptoms and Course.—The effect of mitral disease upon the circulation must, in the main, be the same as that which we have described as occurring in uncompensated aortic valvular disorder.

If, in case of insufficience, a part only of the blood enter the aorta upon systole, the rest regurgitating into the auricle; or, in a case of constriction, if too little of it flow into the ventricle, upon diastole, it is clear that in either case the amount of blood propelled must be smaller than normal, and its flow must be retarded. In like manner the arteries of the aortic circuit contain too little blood, and contract by virtue of their elasticity, while the blood by which they should be filled is overloading the pulmonary system. If the latter be incapable of accommodating all the blood, engorgement of the venous system of the aorta must follow. We have seen that hypertrophy of the left ventricle neutralizes all these circulatory derangements in disease of the aorta. A greater portion of them, but not all, may also be remedied for a time in mitral disease, by hypertrophy of the right ventricle.

The dilated and hypertrophied right heart propels so large a mass of blood, and propels it with so much power into the vessels of the pulmonary circuit, that the blood in the pulmonary veins is subjected to heavy pressure. In consequence of this, to say nothing of the action of the auricle, the blood pours with such force and rapidity into the left ventricle as to completely neutralize the effect of the constriction of the valve. In spite of the constriction, the ventricle receives blood enough; the aortic contents are not lessened, nor is the circulation retarded. In the same way, the fulness and tension of the pulmonary vein prevent any considerable regurgitation into the ventricle, notwithstanding the insufficience of the valve; indeed, as we have seen, the left ventricle is usually both hypertrophied and dilated, so that, in spite of the regurgitation of a considerable amount of blood, it still remains capable of filling the aorta. Retardation of the circulation, with engorgement of the venous system, and a corresponding emptiness of the arteries, is thus averted by a compensating hypertrophy of the right ventricle; but there is one anomaly, which, in aortic disease, is corrected by hypertrophy of the left ventricle, but which hypertrophy of the right ventricle is unable to obviate when the mitral is diseased. This affection is overcharge of the vessels of the pulmonary circuit.

Clinical experience entirely corroborates this physiological, or, rather, physical demonstration. Patients with mitral disease are always short of breath, in consequence of hyperæmia of the lung. As the vessels of the bronchi are less affected than those of the air-cells by this engorgement, the dyspnœa is not always combined with bronchial catarrh; as, however, the bronchial and pulmonary arteries anastomose, nay, as part of the blood of the capillaries and bronchial arteries flows into those of the pulmonary artery, the dyspnœa is generally accompanied by bronchial catarrh. Even at this early stage of the disease, unusual exertion, or other stimulant to the action of the hypertrophied right heart, may cause the death of the patient from acute pulmonary œdema, although such an event is of more common occurrence at a later period, after obstruction of the aortic veins and of the thoracic duct has thinned the serum of the blood.

Patients with insufficience and constriction of the mitral valve often enjoy tolerable health, excepting that they are short of breath, and we should err greatly in supposing that disease of the mitral valve is always accompanied by cyanosis. In constriction of the valve, particularly if combined with insufficience, the compensation soon becomes imperfect. The patients look pale from lack of blood in their arteries; but this derangement of distribution does not cause engorgement of the veins, mainly because most of the blood is collected into the pulmonary circulation.

Sooner or later, the picture changes. Compensating hypertrophy of the right ventricle has its limits, while deformity of the valves grows worse and worse from fresh endocarditis, or else the conditions described in the previous chapter arise, and compensation becomes imperfect. Then the contents of the aorta and its branches diminish more and more, the secretion of urine is lessened, the veins and capillaries become overloaded, the lips and cheeks assume a bluish or even a deep-blue hue. The embarrassed outflow of the cerebral veins creates heaviness in the head, headache, etc. The liver soon becomes enlarged, the patient complains of fulness and oppression in the right hypochondrium; the liver forms a tumor, distinctly demonstrable by percussion and palpation, and which may extend down almost to the navel. Obstruction of the hepatic veins may so increase that the repleted vessels compress the biliary passages, so as to give rise to retention and reabsorption of the bile. The mucous membrane of these passages may also become the seat of a catarrh, and the flow of mucus thus produced may so obstruct the bile-ducts as to cause biliary absorption. A yellow color is thus added to the previous cyanotic aspect, which may impart a greenish tint to the complexion. Chronic gastric and intestinal catarrh arises from obstruction of the gastric and intestinal veins; the hæmorrhoidal veins swell; engorgement of the uterine veins occasions menstrual derangement. Finally, should any considerable congestion of the kidneys set in, there is derangement of the secretion of urine, such as may be produced by ligation of the emulgent veins. The urine is scanty, and contains albumen, blood-corpuscles, and the so-called fibrinous or exudation cylinders, that is, microscopic casts of the urinary tubules, the diagnostic importance of which is to be considered more in detail when we come to study diseases of the kidney.

Venous engorgement, moreover, leads to one of the most important, and, in long-standing cases, one of the most constant symptoms of mitral disease, namely, dropsy. As before observed, an impoverishment of the blood, particularly a diminution of its albumen, contributes essentially to the establishment of transudation of serum. This impoverishment is easily traceable to engorgement. Embarrassment to the outflow from the veins extends itself to the thoracic duct, and obstruction of this duct, of course, impedes the supply of nutritive material to the blood. The dropsy almost always begins in the extremities, generally in the region of the ankles; thence it gradually extends over the thighs, the external genitals, the integuments of the abdomen, and so to the rest of the body. The serous sacs also become the seat of dropsical effusions, producing ascites, hydrothorax, and hydropericardium. Years may elapse after the first appearance of œdema about the ankles, the patient alternately improving and growing worse; his feet now swelling and now growing smaller again, ere the general dropsy is established, of which he, in most cases, ultimately dies. In other cases, he rapidly declines as soon as the first signs of serous effusion show themselves. In many cases an erythema is set up about the genitals, the groins, etc., which is very distressing to the patient, and which, not uncommonly, terminates in diffuse gangrene of the skin. When hydrothorax and hydropericardium develop, his condition grows desperate. The dyspnœa becomes extreme; he can no longer lie down. The serum finally so fills up the air-cells of the lungs, that the blood becomes surcharged with carbonic acid, and his last hours, at least, are relieved by a merciful stupefaction. While the majority of cases thus terminate by dropsy and final œdema of the lung, death takes place, in a smaller number, in consequence of metastases, hæmorrhagic infarction of the lung, or of intercurrent maladies. How much Bright's disease contributes in producing a speedy death is difficult to decide; at all events, whether due to it or not, albuminuria certainly promotes the tendency to dropsy.

*Physical signs of insufficience of the mitral—Inspection and Palpation.*—We often see and feel a strong shock, or even a rise and fall of the thoracic wall over all the region which is in contact with the left ventricle. The apex is displaced outward and somewhat downward. Simultaneously with the shock against the thorax, the epigastrium is also shaken rhythmically. We have considered each of these symptoms while treating of hypertrophy of the right side of the heart, to which they are due. *Percussion* reveals an extension in width of the cardiac dulness. Upon *auscultation*, instead of the first sound, we hear at the apex a murmur, generally somewhat loud, which arises from the irregular vibrations of the valve, which, being roughened and uneven, is in a very unfavorable state to vibrate normally. Sometimes we hear the murmur better, if we listen more above and to the outer side of the apex, as, from hypertrophy of the right heart, the left ventricle of which the apex is formed is, as it were, pushed off from the thoracic wall. As the second sound heard over the ventricle is merely transmitted from the arteries, it presents no abnormity in pure mitral insufficience. Above the aorta, the sounds are feeble; over the pulmonary artery, they are remarkably loud, especially the second, and this intensification, which is still more marked by contrast, is of great diagnostic value. Sometimes, even, we feel a distinct shock at the root of the pulmonary artery, during diastole of the ventricle. Pulsation of the veins, with rhythmical dilatation, does not occur in mitral insufficience, unless complicated by valvular derangement of the tricuspid; although we often may observe a rhythmical undulation of the jugulars, isochronic with systole of the ventricle.

This proceeds from transmission, the strong shock suffered by the tricuspid's being conducted along the column of blood above it, and continues uninterrupted, excepting by the delicate valves, as far as the jugulars. Although the valves in the veins prevent regurgitation of the blood, they cannot check the transmission of a wave of vibration along their contents (*Bamberger*).

*Physical signs of stenosis of the mitral.*—Here, too, inspection and palpation show the signs of excentric hypertrophy of the right side of the heart. The impulse is not usually as strong as it is in insufficience, as the left side of the heart does not take part in the hypertrophy. Besides this, it is much more common in insufficience than in hypertrophy to perceive the *frémissement cataire*, that slight, whizzing sound at the apex, which immediately precedes the beat of the heart, and which ceases suddenly as the beat commences. This phenomenon, the præsystolic purring, is often perceptible through thick clothing, and is so characteristic as in itself almost to suffice to establish the diagnosis of stenosis of the mitral. Upon auscultation we almost always hear a long-drawn murmur at the apex during diastole. Although the blood, as it pours through the normal spacious orifice, occasions no sound, this is by no means the case when it has to be driven forcibly through the narrow passage produced in this disease. The sound is all the louder, the more rapidly the blood pours in, and the rougher and more uneven the surface over which it flows. As a longer time is needed for it to pass through the contracted auriculo-ventricular orifice to fill the ventricle, the murmur heard in mitral stenosis is of longer duration than others, and almost always extends over the whole pause, until cut short, as it were, by the next systolic sound. *Traube* therefore calls a "præsystolic" murmur at the apex a pathognomonic symptom of stenosis of the mitral valve. If the contracted orifice be not also roughened, if the stenosis be moderate, if the volume of the blood be reduced, there may be no sound. In addition, we can, of course, hear the second sound propagated from the arteries, unless the murmur be too loud. Whether we hear the first sound, or a murmur be audible in its stead, depends upon the efficience of the valve. The second sound of the pulmonary artery is naturally considerably intensified.

TREATMENT.—It is not to be supposed that we can cure valvular disease of the mitral by any therapeutical interferenee whatever. We are equally helpless against the consecutive hypertrophy of the right ventricle, which, however, has a beneficial action upon the distribution of the blood. We are, therefore, reduced to a treatment of the more prominent and dangerous of the symptoms.

Hyperæmia of the lung is an inevitable consequence of mitral disease; it cannot be averted nor permanently relieved. We should, therefore, never interfere actively unless it be severe, or unless there be danger of œdema of the lung. This is the more important, as blood-letting, the only active remedy against hyperæmia, although for the time it may ward off the peril, is extremely dangerous for the patient. Perhaps, prior to the bleeding, there may have been no effusion into the subcutaneous areolar tissue. Soon after it the blood will have regained its former volume; but its serum has now become so much attenuated as to transude under a pressure which would not previously have caused transudation. The symptoms of dropsy often first set in immediately after the first phlebotomy. Such "curæ posteriores," however, should not make us hold our hand, if the preservation of life really demand venesection (see chapter on pulmonary hyperæmia and œdema of the lung).

In digitalis we possess a very powerful means of moderating, not only hyperæmia of the lungs, but also engorgement of the aortic venous system which arises in mitral disease. If we can succeed in retarding the action of the heart by means of digitalis, we afford time to the auricle to drive its contents into the ventricle through the contracted passage. Sometimes systole and diastole can be so greatly prolonged (*Traube*) that a pause intervenes between the murmur and the next systolic sound, so that it can no longer be called præsystolic. A marked improvement often accompanies such a result; the breathing grows more free, the swelling of the liver subsides, and the cyanosis and dropsy abate. Latterly, since I have grown bolder in the use of digitalis, and rid myself of the theory of *Traube*, even in cases of insufficience of the mitral, particularly if the heart's action be much accelerated, I have seen the dropsy, cyanosis, and tumefaction of the liver diminish or disappear, while the urine became more copious after the use of an infusion of digitalis. I have come to the conclusion that, by proper administration of this drug, compensation, which is beginning to fail, may, for a time, be reëstablished.

The action of diuretics upon dropsy, resulting from heart-disease, is, at least, a doubtful matter. If digitalis act here as a diuretic, it is probably because it readjusts the circulatory derangement, and thus permits more blood to fill the arteries and thereby affecting the glomeruli of the Malpighian capsules. An agent, intended to relieve suppression of urine, caused by disease of the heart, must either have a special action upon the circulation like digitalis, or it must cause dilatation of the arterioles of the kidney, so that more blood may enter them from the scantily-filled aorta; or else it must so alter the structure of the walls of the renal vessels, as to facilitate the transfusion of liquids through them. True, as long as the class of diuretics has any reputation left, it will be difficult to refrain from prescribing cream of tartar, the alkaline carbonates, squills, etc., when we see the urine daily diminishing, while the serous effusion augments; but, at all events, their action upon the diuresis and dropsy of cardiac disease is inexplicable and remarkably small.

Preparations of iron, on the other hand, are of signal efficacy in dropsy, as is also a nourishment rich in albumen and other protein substances. As already observed, we are totally unable to explain the effect of iron upon the composition of the blood, which consists in an increase in the number of its red corpuscles and of the amount of albumen. However, just as bleeding, by thinning the blood, favors dropsy, so iron and a nitrogenous diet, by rendering the serum more concentrated, have an antihydropic action, and deserve the utmost reliance in treatment of both mitral and aortic disease.

We may afford great assistance, by the institution of a treatment of the symptoms adapted to the phase of the disease, while all exclusive treatment will do harm.

## CHAPTER VIII.

### INSUFFICIENCE OF THE SEMILUNAR VALVES AND CONTRACTION OF THE MOUTH OF THE PULMONARY ARTERY.

As endocarditis scarcely ever attacks the right heart during extrauterine life, and as atheroma of the pulmonary arteries is rare, it is easy to see that valvular deformities, which are almost always the consequence of one or other of these morbid processes, have been met with in the pulmonary artery in but few solitary instances. In these the insufficience depended upon the same causes which occasion valvular disease of the aorta. The few cases of stenosis on record do not always affect the valve ring, some of them arising from annular induration of the conus arteriosus.

The symptoms of valvular insufficience of the pulmonary artery seem to be mainly those of hypertrophy of the right ventricle, just as the excentric hypertrophy of the left ventricle forms the chief sign of corresponding aortic disease. In the cases which have been observed, the quantity of blood in the lungs was not abnormally small, indeed was abnormally great. Dyspnœa, hæmorrhagic infarction, and even consumption of the lungs, followed upon the insufficience. Stricture, at this point, too, seems to be less perfectly neutralized by consecutive hypertrophy, so that cyanosis, dropsy, and other tokens of venous engorgement of the greater circulation soon set in in cases of contraction at the root of the pulmonary artery. Diagnosis of valvular disease of this artery is only possible by means of physical examination; as the functional disturbances, to which the malady gives rise, admit of a too manifold interpretation. In either case, but more especially in insufficience, we find the signs of enlargement of the right heart, so often described; and over the region of the pulmonary artery (that is, over the third left costal cartilage) a murmur during systole is audible in stenosis, while in insufficience it is heard during diastole. These murmurs are produced just as those are which occur in the aorta; they are heard most distinctly over the right ventricle, and over the left upper region of the chest, but are inaudible in the carotids. On account of the extreme rarity of valvular disease at this point, we must employ the utmost caution in diagnosis, and make sure that the murmur heard in the region of the pulmonary is actually loudest at that point, and is not conducted from the aorta.

The treatment can only be symptomatic, and the same rules which we have set forth in the foregoing chapter are applicable here in management of the more threatening manifestations.

## CHAPTER IX.

### INSUFFICIENCE OF THE TRICUSPID, AND STRICTURE OF THE RIGHT OSTIUM ATRIO VENTRICULARE.

A SO-CALLED relative insufficience of the tricuspid used formerly, upon theoretical grounds, to be regarded as a very common form of valvular disease. The ostium was seen to be enormously widened, and it was assumed that the valve was incapable of closure. This relative insufficience, if it ever occurs, is rare. When the ostium dilates, the valve grows in breadth and length, almost always remaining competent to close the widened orifice. Primary and independent disease—thickening, shrinking, etc.—of this valve is also quite rare. It is more common for it to accompany similar disorder of the mitral. *Bamberger*, indeed, regards the combination of mitral and tricuspid deficience as the most frequent of all combinations of valvular defect, and I, too, have repeatedly observed contraction of the tricuspid, with rupture of the chordæ tendineæ, as an accompaniment of severe stricture of the mitral.

In insufficience of the tricuspid, which is in general pure (stenosis being extraordinarily rare), the blood regurgitates into the vena cava during systole of the ventricle; but, as the right ventricle is generally hypertrophied in consequence of mitral disease, this regurgitation takes place with great violence. The vena cava and the jugulars become enormously dilated. The valves of the jugular, which, if its calibre were normal, would set a limit to the regurgitation, become insufficient from dilatation, and it is transmitted as far as the vessels of the neck. *Real pulsation of the dilated jugulars, perceptible both to touch and sight, is a pathognostic symptom of insufficience of the tricuspid.* Besides this, we hear a distinct systolic murmur at the lower part of the sternum, which, in conjunction with the venous pulsation, makes the diagnosis certain; but here also we must make sure that the murmur is really strongest at this point, and is not conducted thither from the aorta.

As insufficience of the tricuspid causes the most intense engorgement of the veins of the aortic circulation, so, of all valvular disorders, this leads most rapidly to cyanosis and dropsy.*

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* When one valvular defect complicates another, the symptoms of the former one are modified. The modifications vary according as the complication has a similar or an opposite effect upon the circulation; and according as one or other defect predominates. The signs of complex valvular disease may easily be deduced from the analysis of the foregoing chapters.

# CHAPTER X.

## DEGENERATION OF THE SUBSTANCE OF THE HEART, GROWTHS, PARASITES.

ETIOLOGY AND PATHOLOGICAL ANATOMY.—1. An abnormal softness, relaxation, and flabbiness of the substance of the heart, imparting to it "a parboiled look" (*Rokitansky*), are not uncommon in the bodies of those who have died of typhus, septicæmia, puerperal fever, etc. No important alteration can be detected in its structure, and we must beware of mistaking the relaxation resulting from decomposition for that which has taken place during life. The state of other organs must form our criterion in this case.

2. *Fatty Heart* must be regarded as of two kinds:

*a.* Increase of the amount of fat normally found upon the surface of the heart.

*b.* Fatty metamorphosis of the primitive fasciculi of the muscular substance.

In the former we find a layer of fat, half an inch thick, covering the heart, particularly along the course of the coronary arteries, upon its edges, and in the sulcus between the two chambers. Beneath this fatty layer the muscle is either normal or has undergone atrophy and thinning from pressure of the superimposed fat. In many cases, atrophy of the muscle occurs while the fat-tissue is forming, and without the growth of the latter having become very remarkable. This growth, then, takes place at the expense of the substance of the heart, so that a cardiac wall of normal thickness may at last consist only of adipose tissue. This excessive production of fat in the heart often accompanies general obesity, especially that of advanced age, and in subjects otherwise healthy. It is also seen, however, in cancer, and in other, cachexia, and especially among drunkards.

*Fatty metamorphosis of the primitive fasciculi* consists in conversion of the fibrillæ into fat-granules, which gradually fill the entire sarcolemma, and afterward combine to form large drops. Thus the substance of the heart becomes discolored, and is converted into a pale-yellowish mass which tears readily. Sometimes the metamorphosis pervades large tracts of the organ, while in other cases particular parts only are affected, as, for instance, the papillary muscles. Accompanied by arcus senilis, and fatty degeneration of the arteries, it often forms one of the signs of marasmus senilis, or of other marasmic states, which arise in cancer, Bright's disease, etc. Ossification of the coronary arteries, pressure of pericardial exudation, or even that accumulation of fat upon the surface of the heart just mentioned, may give rise to fatty degeneration of its walls. Finally, in many cases of valvular disease, with consecutive hypertrophy, a partial fatty metamorphosis takes place.

The origin of this "spurious hypertrophy" is somewhat obscure, and almost without parallel; while fatty degeneration of the cardiac muscles, in consequence of defective nutrition or pressure, etc., finds many analogies in the metamorphosis of other organs whose nutrition is impaired.

3. *Amyloid degeneration*, according to *Rokitansky*, occurs especially in the hypertrophied right side of the heart, causing its cut surface to resemble that of a piece of bacon, and occasioning great rigidity of its wall. The sarcolemma is filled up by nodules, which glitter dimly and show the peculiar reaction of amyloid degeneration, turning blue upon application of a dilute solution of iodine and weakened sulphuric acid.

4. *Cancer* is very rare in the heart, occurring only in general cancerous infection, or by extension from the mediastinum or pericardium. It forms circumscribed tumors, usually of the medullary, or else of the melanotic kind, which project either inward or outward, and may sprout into the cavity of the organ. In other cases, especially where propagated from cancer of neighboring parts, wide tracts of the substance of the hearts become transformed into cancer (infiltrated cancer, see cancer of the lungs).

5. *Tubercles* scarcely ever occur in the heart. Yellow, cheesy nodules, sometimes found embedded in its walls, are not to be regarded as tubercles, and shall be accounted for when we come to treat of pericarditis.

6. *Parasites.*—The cysticercus has been found in the heart, enormous numbers of them existing at the same time in other muscles of the body. The echinococcus has also been met with.

SYMPTOMS AND COURSE.—Relaxation of the cardiac substance, after typhus, exanthematic disease, etc., of course reduces the efficience of the organ, and is very apt to occasion dilatation. It is only in the latter case that we are able to recognize it with certainty. If, after an attack of some exhausting disease, we find that the impulse of the heart is extraordinarily feeble, the area of cardiac dulness having increased, we may also attribute the small pulse, the dropsical aspect, the spontaneous coagula in the veins, in part, at least, to the structural changes which the substance of the organ has undergone.*

If the existence of dilatation cannot be proved, we must remain in doubt as to whether the retarded circulation and the scanty arterial supply be due to general exhaustion or to relaxation of the heart.

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* In such instances cyanosis is rare, as the volume of the blood is reduced and its quality is very poor; so that, even though it were to overfill the veins, it would not give rise to the bluish complexion.

Growth of fat about the heart plays an important *rôle* among the people, in accounting for shortness of breath and other troubles arising among fat, pot-bellied individuals. Unless the accumulation cause atrophy of the muscular substance, which is by no means frequent, it it does not seem to occasion any functional disturbance whatever. Should atrophy result, the symptoms already mentioned (see atrophy of heart) will arise.

Fatty degeneration of the cardiac substance, like simple relaxation of it, depresses the action of the organ, and in like manner, when it affects the whole heart, occasions dilatation. All the circulatory disturbance which we have so repeatedly described may ensue from fatty degeneration. We find a feeble heart-shock, a small and remarkably slow pulse, a tendency to faintness, from an imperfect supply of blood to the brain. If the volume of the blood be not diminished, that is, if the degeneration depend upon local rather than upon general nutritive disorder, there may also be cyanosis and intense dropsy. In the latter case, when the disease is usually combined with other affections capable of reducing the propulsive power of the heart (such as pericardial exudations, induration and thickening of the pericardium, etc.), it is difficult to determine what part is taken by these disorders, and what the degeneration plays in producing the train of symptoms; so that a positive diagnosis is impossible in most instances. The same thing holds good with regard to the transition from genuine to spurious hypertrophy, through fatty degeneration of the muscular fibres. If the impulse and contractile force of a hypertrophied heart become manifestly weaker, or if compensation for an imperfect valve begin to fail, we may assume that the change from true to false hypertrophy has taken place. Fatty metamorphosis of the papillary muscles is also to be reckoned among the possible consequences of insufficience of the mitral or tricuspid.

Of rupture of the heart, as a result of fatty degeneration, we shall treat hereafter.

As for amyloid degeneration of the substance of the heart, evidence, often easy to obtain, of existence of the disease in the liver, spleen, or kidney, affords our only but uncertain clew as to its presence in the heart; and we can never do more than vaguely suspect it.

Cancer, tubercles, and parasites of the heart, also have the effect of depressing its action, but their diagnosis is almost always impossible.

TREATMENT.—If the heart be relaxed by debility following acute disease, the remedies so often named are called for which have the effect of improving nutrition, together with mild stimulants.

Persons in whom a general obesity has developed, through luxurious living, and in whom an accumulation of fat may also be suspected about the heart, should be sent to Karlsbad, Marienbad, etc. It is an indisputable fact that, during treatment at these baths, the fat decreases and the garments of patients grow too loose for them, although we have no better physiological explanation of the circumstance than a somewhat feeble hypothesis.

In true fatty degeneration of the heart we must confine ourselves to a treatment of symptoms, and, if it form one of the accompaniments of general marasmus, we should prescribe a generous diet and corresponding medicines. We may, perhaps, succeed in restraining the progress of the malady, if we do not entirely allay it.

Treatment of amyloid degeneration, cancer, tubercle, and parasites, is out of the question, as the diseases are never recognizable.

## CHAPTER XI.

### RUPTURE OF THE HEART.

We refer exclusively to the so-called spontaneous ruptures, and shall not allude to traumatic solutions of continuity of the heart. A healthy heart never bursts, in spite of the greatest strain. If the organ be diseased, strains of any kind may, no doubt, aid in causing its rupture. The most frequent cause of rupture is fatty degeneration; more rarely, myocarditis, cardiac abscess, and acute and chronic cardiac aneurism. As all of these affections usually arise in the left side of the organ, rupture nearly always occurs there also. Upon autopsy, we find the pericardium distended by blood, and, if fatty metamorphosis have occasioned the rupture, an irregular but outwardly smooth rent, of variable length, is found; while at a deeper point the flesh is torn asunder and mangled. The rent is occasionally filled throughout by coagula; again more than one rupture is found.

Sometimes the heart bursts during some unusual exertion, or it may give way without any apparent cause, and death usually ensues suddenly with the symptoms of internal hæmorrhage. The pressure of the extravasated blood, however, also seems to have some effect in promoting speedy death. In rare instances the rupture has been preceded by a brief period of violent pain under the sternum, shooting toward the left shoulder and along the arm. In cases equally rare, patients have survived rupture of the heart for several hours. This seems to happen when the extravasation consists, at first, of a mere filtration of the blood through the broken-down, disintegrated cardiac wall, the rent gradually growing larger. Symptoms then appear of a less active haemorrhage; and it is sometimes possible to make out the physical signs which mark the progress of the flow into the pericardium.

## CHAPTER XII.

### FIBRINOUS DEPOSITS IN THE HEART.

We seldom dissect a body without finding in its heart a clot of fibrin, especially in the right side of the organ. Sometimes the clot is yellow, consisting entirely of fibrin, which has separated from the red portion of the blood; sometimes it contains red corpuscles, and is more or less colored. Their tenacity is variable, and they are usually entangled among the trabeculæ, but may easily be separated from the endocardium. In the bodies of persons who have died of pneumonia, or other disease in which the fibrin of the blood is increased in quantity, these coagula are found especially large, and, if removed from the heart, long clots, forming prolongations into the arteries, are drawn after them. These fibrinous clots, or false polypi of the heart, have formed after death, or during the period of dissolution. The more protracted the latter, so much the longer is the blood, as it were, whipped up in the heart, so much the more completely is the fibrin separated from the red blood, and so much the more colorless and intimately entangled in the trabeculæ is the resulting clot. In other instances, the coagula seem to have formed some time before death. The fibrin has lost the elasticity and glitter of fresh fibrin, and is firmer, drier, and yellower. The clots are tightly adherent to the endocardium, and we sometimes find their interior decomposed into a puruloid, yellowish, or brownish-red emulsion, or converted into a yellowish, cheesy mass. No real pus is formed, but a mess of *débris*, in which colorless blood-corpuscles must not be mistaken for pus-corpuscles.

Sometimes we find fibrinous deposits in the heart, in the form of rounded, wedge-shaped masses, in size from that of a millet-seed to that of a nut (*Laennec's* végétations globuleuse). If we examine their mode of attachment more attentively, we perceive numerous roots proceeding from the spherical vegetations, which are prolonged deeply into and entangled among the meshes of the trabeculæ. The source of the vegetations is to be sought there; the spherical form is the result of subsequent deposit upon the clots first formed. Here, too, the softening just mentioned is found sometimes in the interior of the coagulum, so that at last they may assume the appearance of sacs, with thin walls and puruloid contents. We have already spoken of the deposits which form upon roughened places on the endocardium, from endocarditis, acute or chronic aneurism of the heart, and valvular disease.

The coagula which form prior to death are mainly the result of the feeble manner in which the heart contracts. Hence, they are commonly found among marasmic subjects, and in persons who have degeneration of the heart. Their points of origin are always the shallow recesses between the trabeculæ, which readily dilate when the heart is relaxed or softened, so that, if its contractions be incomplete, the blood in them stagnates and coagulates. In very rare instances an embolus may, perhaps, form the nucleus of a clot.

When coagula form in the heart during the death-throes, they may, no doubt, occasion some obstruction to the circulation, but it is impossible to know how much of the feebleness of the circulation is due to the palsy of the heart, and how much to obstruction of the orifices by clots. Even if the clots produce murmurs, they cannot be distinguished from the murmurs caused by irregular and imperfect action of the heart. This is true also for the clots which form prior to the death-agony, as they, too, form in case of feeble cardiac action and impeded circulation.

## CHAPTER XIII.

### CONGENITAL ANOMALIES OF THE HEART.

ETIOLOGY.—The majority of congenital defects of the heart are due either to arrest of development (the organ remaining in a condition which was normal during fœtal life), or else to fœtal endocarditis or myocarditis. We are unacquainted with the causes of this arrest of development, as well as with the causes of the fœtal inflammation.

Prominently in the former class of congenital deformities of the heart stands incompleteness of the septa; in the second class, the indurated strictures produced by inflammation, and congenital stricture and insufficience of the valves, are the most important. The latter occur generally in the right heart, which, after birth, is very rarely attacked by endocarditis or myocarditis.

The causes of congenital malformation in position of the heart may be similarly classified. Sometimes they are to be regarded as cases of arrested development, the ribs, the sternum, and the clavicles being imperfectly formed, so that a greater or smaller portion of the heart is covered by soft parts alone. In other cases they depend upon inflammation during fœtal life, which has given rise to adhesions with neighboring organs.

The pathogeny of dextro-cardia, in which the heart lies on the right side of the thorax, the liver generally occupying the left hypochondrium, and the spleen the right, etc., is altogether obscure.

ANATOMICAL APPEARANCES.—Congenital deformities of the heart, which are incompatible with life, and which cause children to die either immediately, or else very soon after birth, belong rather to the province of pathological anatomy than to that of special pathology and therapeutics. Entire absence of the heart, or of one of its chambers, is one of these. In anomalies which permit the continuance of life, even for a short time, we generally find every part of the organ represented, although some portions of it are only rudimentary. In most instances the aorta or the pulmonary artery is stunted, or quite undeveloped. If the pulmonary artery be deficient, the blood pours from the right heart directly into the left, as such cases are always combined with imperfection of the septa. The aorta then supplies the lungs with blood through the dilated bronchial arteries, or through the ductus Botalli, in which it sets up a current counter to the fœtal blood-stream. If the aorta be contracted or closed immediately above the opening of the ductus Botalli, it then can only supply the head and upper extremities, while the pulmonary artery conveys blood to the lower half of the body through the ductus Botalli. If the aorta be closed at its origin, the blood which comes to the left heart passes directly through the open septum into the right heart, the pulmonary artery then furnishing blood to the whole aortic system. When the septum between the ventricles is imperfect, it may seem as if the aorta and pulmonary arteries sprang from both of them. If the septum stand too far to the right or left, the right or left ventricle will be too large, and both arterial trunks will originate from it, while the stunted ventricle has to discharge its blood into it through the open septum. In very rare instances, the aorta has been found to spring from the right, the pulmonary artery from the left ventricles. There also are anomalies of the veins discharging into the heart, to describe which, however, would carry us too far.

Insufficience and stenosis of the orifices, and cicatricial strictures of the heart consequent upon fœtal endocarditis and myocarditis, differ from those acquired after birth, in that their situation is in the right heart. Valvular disease is more common at the pulmonary valves than at the tricuspid. In these cases, too, the septum is not closed, so that transfusion of the blood takes place from one side of the heart to the other.

Defects may exist in the septa of greater or less extent, but they do not afford complications and results like those just described, which are of far less importance, and, indeed, may be without any material influence whatever upon the circulation, and, hence, are to be regarded as independent, and proceeding from arrested formation due to unknown causes. In particular, we very often make *post-mortem* discovery of slit-like openings, or even great holes, in the foramen ovale, which openings have never occasioned any symptoms whatever during life. In the septum of the ventricles, likewise, especially at a point at the upper end, which normally is very thin, it is not uncommon to find imperfections, of more or less magnitude, which have never given rise to any inconvenience.

In the higher grades of ektopia, in which a greater part of the wall of the chest or belly is wanting, the heart lying in the abdomen or upon the neck, continuance of life is impossible. There are persons alive, however, with smaller imperfections of the bony thorax, fissures in the sternum, etc., and who even have attained an advanced age. In such cases the deformity is covered by the skin, and the subject suffers little inconvenience.

Symptoms and Course.—If we keep the effect in view, which congenital malformations of the heart exert upon the circulation, turning first to the most frequent and important of them, that namely, in which the aorta or pulmonary artery with its ventricles is undeveloped, so that the blood passes through the open septum, from one side of the heart to the other, and is carried into the body through the more perfect trunk alone, it will be apparent that the following derangements in the distribution of the blood must occur.

First, the current of the blood-stream is greatly retarded, and hence the blood, tarrying long in the body and rarely returning to the lungs, is overloaded with carbonic acid, and assumes an intensely venous character. Ceteris paribus, the rapidity of the circulation depends upon the volume of blood set in motion by every heart-beat. If the aorta or pulmonary artery be missing, if but one outlet from the heart remain, then, notwithstanding hypertrophy of the ventricle, the volume of blood set in motion must be far too small. The retardation of the circulation thus resulting sufficiently explains a series of symptoms observable in congenital imperfections of the heart—the lassitude, languor, intellectual apathy, depressed spirits, and, above all, the low temperature of the body.

If, however, the supply of blood to the greater and lesser circulation be furnished by only one of the ventricles, it must follow that the arteries are very scantily filled, and that venous engorgement of great intensity arises, as in all other cases, where the arteries have not their due supply. Accordingly, we find the pulse small, the breathing very short, and, above all, we observe cyanosis, the symptom which we have so often designated as the characteristic one of overloading of the veins. Since cyanosis arises here, as it does elsewhere, from obstruction to the course of the blood through the capillaries and veins, the extreme intensity which it here exhibits must be owing to some other cause, which is to be sought in the excessively dark hue of the blood.

This blackness of the blood is not in itself capable of producing cyanosis (as is shown by a case, related by *Breschet*, in which the color of a left arm was perfectly normal, although it contained none save venous blood, the left subclavian artery springing from the pulmonary artery). It cannot be denied, however, that where venous engorgement exists, the degree of cyanosis depends upon the blackness or redness of the blood. Thin-blooded people never exhibit much cyanosis. In cases of congenital imperfection of the heart, in which the pulmonary artery is undeveloped, the dark color of the blood may be ascribed to the mixture of venous and arterial blood which then takes place. In the converse cases, wherein the arterial part of the blood is thrown into the venous portion, the cause of the darkness of hue cannot be found in the mixture, but is to be attributed rather to the extreme retardation of the current.*

Induration and stricture at the conus arteriosus of the right heart, and the extensive valvular deformities which affect the pulmonary artery, have an effect quite similar to that of arrested development of the arterial trunks, especially as in these cases, too, the septa remain open.

The deep-blue color of the skin, particularly that of the face, the lips, cheeks, and the tips of the fingers and toes, is the most conspicuous symptom of congenital deformity of the heart. The collection of venous blood in certain parts also causes their enlargement, due (as has been ascertained by the careful observation of *Foerster*) to serous infiltration, moderate thickening, and hypertrophy. The nose becomes bulbous, the bluish lips swollen, and the terminal phalanges of the fingers and toes so much thickened as to look like the knobs of drumsticks. The nails are wide and arched.

Most patients have puny frames with long limbs, and show great tendency to profuse hæmorrhage. They are susceptible to cold; are sluggish, languid, and irritable. They often have imperfect development of the genitals and feeble sexual power. They suffer attacks of palpitation, oppression, and syncope, and rarely attain the age of forty or fifty years. They nearly always die early of intercurrent disease, which they are ill able to resist, or else they perish from œdema of the lung, dropsy, etc.

It is remarkable that sometimes the cyanosis and functional disturbance just described do not manifest themselves until the period of puberty. It may be that, for a time, compensation for the congenital deformity is effected by consecutive hypertrophy, but that insufficiency of the heart only appears after the development and growth of the body and the increase of the volume of the blood have advanced so fast that the puny heart can no longer keep pace with it. Physical examination is of little value in the diagnosis, owing to the great diversity of form which such malformations assume. The impulse of the heart is usually strengthened and extended, the dulness greater; we feel the *frémissement cataire* and hear confused murmurs. In other cases the heart-sounds are normal.

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* That the great cyanosis of persons with congenital malformation of the heart is due to an especial cause, becomes evident from the fact that individuals with congenital cyanosis do not become dropsical nearly so soon as those suffering from acquired cyanosis. This would not be the case if the cyanosis were due to venous engorgement alone.

The ancient assumption, that imperfection in the septa caused cyanosis, is erroneous. This defect, alone, never occasions blueness, etc., but is a harmless anomaly, which gives no evidence of its existence during life.

TREATMENT.—Treatment of congenital deformity of the heart must, of course, be purely symptomatic, and confined to combating the more dangerous manifestations. The same rules hold good here which we have laid down for the management of acquired disease of the heart.

## CHAPTER XIV.

### NEUROSES OF THE HEART.

ETIOLOGY.—There are a number of influences which tend to modify the functional energy of the healthy human heart, as well as the number of its beats. We may assume that the greater force and frequence of the heart-beat, caused by mental or bodily excitement, or by the use of ardent spirits, are not the effect of any structural change in the muscles of the organ, but rather are due to a perversion of its innervation. The term neurosis of the heart, however, is not applied to functional derangements proceeding from causes of this nature, but only to those forms of perversion of its action or abnormity of its sensation which, without depending upon any structural change, arise either without preceptible cause or else upon occasions which, in most persons, would not give rise to any functional disturbance. Under this head stand the so-called nervous palpitation and the train of symptoms known as angina pectoris. The character of these two affections, particularly the paroxysms and the free intervals observed in their course, entitle us in some measure to count them among the neuroses of motion and sensibility. It would be somewhat rash, however, to ascribe them as yet to any particular class of these complaints, as long as our knowledge regarding the influence of the cardiac nerves upon the function of the heart remains in its present imperfect state.

The nerves of the heart consist of branches of the par vagum and sympathetic, and, besides these, it has its peculiar ganglia. If we separate the organ from the nerves, and cut it out of the body, it still will continue to contract rhythmically for some time; and, even after ceasing to beat, will recommence, if we inject blood into the coronary arteries, or supply it with oxygen. Rhythmical contraction, then, is not dependent upon the pneumogastric or sympathetic nerves, but seems to be brought about solely by the cardiac ganglia, although this too has been doubted. As to the effect of the pneumogastric upon the beat of the heart, we know that irritation of this nerve retards its action, while section accelerates it, so that we may regard the nerve as a moderator of the activity of the heart. We know but little in this respect, and nothing of certainty, concerning the influence of the sympathetic nerve.

It would, therefore, seem rash to count *palpitation* among the hypercineses, a condition due to extreme excitement of the motor nerves, as has been done by *Romberg*, and more lately by *Bamberger*. It is quite as possible that palpitation of the heart might proceed from reduced energy of the pneumogastric as from over-excitement of the sympathetic or cardiac ganglia. Besides, in many cases of nervous palpitation, an increased force of the heart's action cannot be observed, the symptom being merely subjective, and perceptible only to the patient. Such cases should more properly come under the head of hyperæsthesiæ, and be regarded as an extreme excitement of the sensory nerves of the organs.*

*Romberg* defines *angina pectoris* as hyperæsthesia of the cardiac plexus. *Bamberger* calls it a hypercinesis with hyperæsthesia. The cardiac plexus is assumed to be the source of the pain; but this, too, must be pronounced a matter of theory only. At all events, the pain which attends this "cardiac neuralgia" extends with great intensity along the brachial plexus.

Ignorant as we are regarding the pathogeny of neuroses of the heart, we still have some idea as to their cause. Nervous palpitation is principally seen in anæmic subjects, and is one of the most constant manifestations of chlorosis. Next in frequence, we find it in derangement of the sexual system, not only among females, where it plays an important part in hysteria, but also among males addicted to venereal excess, above all, among onanists. Palpitation is also common in hypochondriasis. As a striking instance of this hypochondriac palpitation, *Romberg* relates the example of Peter Frank, who, while engaged in study of disease of the heart, thought himself suffering from aneurism. We very often see palpitation accompany rapid growth about the time of puberty. It affects other persons, in whom no definite exciting cause can be discovered.

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* We should be equally warranted in calling every fainting-fit an acinesis of the heart. When a person swoons from psychical or other causes, the scene always commences by a depression of the heart's action, smallness of the pulse, and pallor of the skin; and it is not until then that the consequences of diminution of the supply of arterial blood to the brain, loss of consciousness, etc., appear. It is quite the same in a too-protracted inhalation of chloroform or ether.

*Angina pectoris* is found almost exclusively in persons suffering from organic disease of the heart. Either ossification of the coronary arteries, valvular defects, hypertrophy, degeneration, or aneurism of the aorta, has been found upon autopsy of most persons who have been afflicted in this manner. Nevertheless, we cannot regard angina pectoris as indicative of any of these lesions. Not one of them is constant; and the malady always takes the same form while the structural alterations differ most widely. It is always marked by paroxysm and intervals of immunity, so that we are forced to set it down as a nervous disorder of the heart, to which organic changes merely afford a predisposition. In rare instances it has occurred where no organic disease existed, particularly in old and obese persons, males being affected oftener than females.

SYMPTOMS AND COURSE.—Nervous palpitation is characterized by an accelerated and sometimes unrhythmical beating of the heart, accompanied usually by a feeling of dread and dyspnœa. The impulse is generally short and bounding; in many cases without perceptible increase of force, and in others so violent as to shake the hand at each stroke. Even in the latter cases the subjective feeling of palpitation experienced by the patient is greater than what the apparent force of impulse would lead us to expect. The pulse and aspect of the patient are not always the same. Sometimes the pulse is full, and the face red; sometimes it is small and intermitting, and the countenance is pale, apparently as if the beats lacked energy, or as though they were of too brief duration effectually to fill the arteries. The length of an attack of this kind varies, lasting from twenty minutes to an hour or more. It is not unfrequently accompanied by nervous derangement of other kinds, dizziness, buzzing in the ears, trembling, etc. Its termination may be sudden or gradual, the action of the heart returning to its normal condition, and weeks or months may pass without the occurrence of a new attack, while in other cases the seizure recurs at very short intervals.

The intermission and recurrence of the paroxysms without known cause, their appearance under conditions which do not, as a rule, give rise to exaggerated action of the heart, its association with other nervous disorders, and, above all, the results of physical exploration, will serve to prevent error, and yet the disease is not always easy of recognition. If the exciting cause be obvious and amenable to treatment, the malady will disappear sooner or later. This is especially the case with chlorotic girls, hysteric women with curable disease of the womb, and even in palpitation induced by excess in venery. At other times it is extremely obstinate, and persists throughout life.

During the intermission, physical exploration reveals nothing anomalous; during the paroxysm, we often hear abnormal murmurs attributable to unnatural tension of the valves and arterial walls.

In angina pectoris, the patient suddenly experiences beneath the sternum a feeling of strangulation and pain, which almost always shoots in the direction of the left arm, less frequently toward the right. This is accompanied by a distressing feeling of dread and sense of impending dissolution. The sufferer imagines that he cannot breathe; but, if forced to do so, succeeds in making a deep inspiration. He does not dare to speak, but groans and sighs. If the attack come upon him while walking, he stands still, seeking a support, and clasping his breast. The hands are cool, the countenance pale, the features perturbed. After the lapse of a few minutes, or in a quarter or half an hour, the paroxysm gradually abates, nearly always with eructations of gas. These attacks are repeated at first with long intervals; afterward they become so frequent as to be of almost daily occurrence. As an exciting cause, mental emotion seems to be the most common agent; physical exertion and error of diet produce it more rarely. Between the attacks health may seem unimpaired, while in other cases evidence of serious disease of the heart may be detected.

TREATMENT.—Treatment of nervous palpitation demands, above all else, the removal of every recognizable and remediable predisposing cause. In chlorotic or anæmic subjects, the preparations of iron often render signal service. Hysterical palpitation may require the application of leeches to the os uteri, and of lunar caustic to the orifice; a treatment which, as we shall see in the proper chapter, will often effect a cure in a case previously hopeless. Hypochondriacs, with varicosities of the anus, if affected by palpitation, often find great relief from the application of leeches to the fundament. Fuller details of the appropriate remedies in this affection would occupy us too long, as it would include the treatment of all the maladies of which palpitation is an accompaniment. Patients, in whom no special cause for the disease can be found, should be ordered to bathe in cold water, be sent into the country, made to travel, and forbidden all over-exertion and luxurious living.

During the attack, the effervescent powders, vegetable and mineral acids, cream of tartar, "eau sucré," enjoy a certain reputation. It would be foolish to carry one's skepticism so far as to slight these medicines, because they are superfluous and impotent; as it is, the mental preoccupation afforded by the preparation of an effervescent powder, etc., is often of the greatest relief to any person afflicted with palpitation, and even shortens the paroxysms. The application of cold over the heart seems to be of decided efficacy in abbreviating the fits. The nervines, tincture of castor, tinctura valerianæ ætherea, often have the same effect. On the contrary, narcotics, especially digitalis, if used, must always be employed with the greatest caution in cases of nervous palpitation, in the narrow acceptation of the term which we employ.

It is doubtful whether it be in our power to relieve paroxysms of angina pectoris by means of any medication; but, after having once witnessed the impatient and hurried clutch of the sufferer for his medicine-glass, as the attack comes on, the physician will readily acknowledge that the "*laisser aller*" mode of treatment is sheer cruelty. *Romberg* advises the inhalation of sulphuric and acetic ether, a couple of teaspoonsful of it being poured into a saucer, and its edge held to the mouth of the patient while it evaporates. Complete narcosis must not be permitted. In these cases, too, I have seen decided abbreviation of the fit by the use of tincture of valerian and castor ætherea. Opiates, and other narcotics, are to be avoided. During the intervals, treatment must be limited to combating whatever recognizable and remediable predisposing cause may exist. Fontanelles, setons, etc., much as they have been made use of, ought not to be employed.

## CHAPTER XV.

### BASEDOW'S DISEASE.

The term *Basedow's* disease is applied to a train of symptoms of tolerably frequent occurrence, consisting of a subjective sense of palpitation, accompanied by acceleration of the action of the heart, beating of the veins of the neck and head, swelling of the thyroid gland, and exophthalmos. This peculiar series of symptoms is sometimes seen in patients with valvular disease of the heart, but is more frequently observed in persons free from any organic cardiac disease. The tumefaction of the thyroid body, which is not often very large, arises partially from dilatation of its vessels, and in part from infiltration of its tissues with serum, and from simple hyperplasia. More rarely cysts, with serous or colloid contents, are found in the gland. The swelling of the intra-orbital fat, which is the cause of the exophthalmos, seems in most cases to be due to hyperæmia and œdema, or to simple hyperplasia of the adipose tissue; since, when recovery takes place, the prominence of the eyes subsides as completely as do the thyroid enlargement and the disturbance of circulation.

That this is not a mere coincidence of morbid conditions, and that it is fully entitled to be regarded as a separate and distinct disease, is evident from the circumstance already alluded to, that the changes in the thyroid and eyes, not only appear simultaneously with the derangement of the circulation, but that they also subside together.

In seeking for a common source to which the individual symptoms of *Basedow's* disease may be attributed, the idea of a derangement of innervation of the vascular walls naturally suggests itself. Palsy of the vaso-motor nerves fully accounts for the dilatation and increased pulsation of the carotids and thyroid arteries, as well as for the œdematous swelling of the thyroid gland and intra-orbital fat. The subject of the innervation of the heart, indeed, is by no means satisfactorily understood, in spite of the labor expended upon the subject; yet it is perfectly supposable that variation in the degree of fulness of the blood-vessels, which traverse the substance of this organ, may have an important influence upon its function; and that palsy of the vaso-motor nerves of the cardiac vessels will cause them to dilate, thus augmenting the supply of blood to the cardiac muscles, and producing essential modification of the heart's action. We have no hesitation in declaring our belief that the probable cause of the symptoms of *Basedow's* disease consists in a subparalytic state of the vessels of the muscles of the heart. At the same time we deem it rash, or at least premature, to ascribe such palsy of the vascular walls to coarse structural changes of the cervical ganglia of the sympathetic nerve. Apart from the fact that the lesions of the ganglia in some cases are entirely different from those found in others, and that in other instances again, in spite of the most careful search, no lesion whatever has been found in the ganglia, it is improbable that the nervous disorder of the vascular wall should depend upon coarse and palpable alterations of texture of the nerve-fibres and ganglion-cells, simply because such nervous derangement often subsides entirely.

*Basedow's* disease is far more common among women than among men; menstrual disorder, or perhaps the lack of red corpuscles in the blood, which so often accompanies such disorder, also seems to have some part in its production; but it is altogether inadmissible to regard such disease of the vaso-motor nerves as a mere part of that widespread disorder of innervation, which occurs in hysteria, and to attribute the relaxed state of the vessels to faulty nutrition, either of the vessels or of their nerves, proceeding from the want of red corpuscles in the blood. Indeed, *Basedow's* disease is not especially prevalent in cases of severe hysteria or intense chlorosis, and in some cases even appears in persons free from both menstrual disturbance and impoverishment of the blood. Men who are affected by this malady are usually somewhat advanced in life, while among women it generally appears during youth.

The patients generally have long suffered from palpitation, with a remarkable frequence of the pulse, which sometimes rises as high as 120 or 140 beats, when the patient and his friends become aware that his eyes are more prominent than formerly, and that the neck is enlarged. If the hand be laid upon the thyroid gland, or the stethoscope be applied to it, a remarkable rustling is perceptible both to ear and touch. Sometimes a blowing sound is also heard at the heart. Generally these sounds are easily recognizable as "blood-murmurs," as there is no secondary dilatation, nor hypertrophy of the organ, without which it is impossible to ascribe a false murmur to valvular derangement. At a more aggravated stage of the malady, the prominence of the eye-balls increases to such an extent as to render the eyelids incapable of covering the eyes completely. This inability to close the eyes may have the most disastrous consequences. In some instances, infiltration, abscess, and perforation of the cornea, and even complete destruction of the eye, have been known to follow. Such accidents are no doubt due in a great degree to a want of proper covering, and lubrication of the bulb, although it would seem that the graver degree of destruction does not occur until after the establishment of a certain amount of anæsthesia of the cornea (attributable to strain upon the ciliary nerves), rendering the eye incapable of protecting itself properly. Sometimes the motion of the bulb is embarrassed, probably in consequence of palsy of the ocular muscles, resulting from stretching; but, excepting the affections of the cornea above alluded to, there is scarcely ever any other derangement of vision. *Graefe* speaks of a spasmodic contraction of the levatores palpebræ superioris as a very characteristic symptom which sometimes precedes the exophthalmos. It becomes recognized by the hesitating and imperfect manner in which the upper-lid is depressed when the eye is made to look downward. In severe cases, the pulsation of the thyroid and carotids is so marked as to be apparent to the eye even at a distance. Most patients complain of oppression; some of dizziness and headache, and of other irregular symptoms.

Generally the disease drags on for months and years. Instances when its course has been acute and rapid are exceptional. If the result is to be unfavorable, it is generally on account of a gradual dilatation of the heart with diminution of its functional power. The patient becomes cyanotic, and dropsical, the obstruction in the veins of the pulmonary circulation gives rise to extreme dyspnœa, and at last to œdema of the lungs. More rarely death takes place with cerebral symptoms, or in consequence of intercurrent disease. An improvement terminating in complete restoration is by no means an uncommon occurrence. Indeed, recovery is a much more common ending of the disorder than is death.

*Basedow's* disease often recovers under a treatment consisting in a strengthening diet, and in the use of iron. The secale cornutum has also been prescribed as having a reputed power of causing contraction of the walls of blood-vessels, and a reduction of their calibre.

Whether the improvement be really in consequence of this mode of treatment, or not, remains a matter of doubt. At all events, it would be well to try this remedy, or some similar one, and for the time to disregard the application of a constant and induced current applied to the cervical portion of the sympathetic, which has been proposed upon purely hypothetical grounds.

When the exophthalmos is very great, *Graefe* advises the use of a light bandage, and in extreme cases the diminution of the opening of the eyelids by means of a surgical operation.

# SECTION II.

## *DISEASES OF THE PERICARDIUM.*

## CHAPTER I.

### PERICARDITIS.

Etiology.—With regard to the pathogeny of pericarditis, we may refer to what we have already said concerning its kindred affection, pleuritis. In many cases, where the disease is partial, the inflammatory derangement of nutrition is not such as to produce interstitial exudation and effusion into the sac; but a proliferation of the pericardium takes place, so that its normal tissue forms offshoots, and becomes thickened. Thus the so-called tendinous spots, etc. (*Sehnenflecke*) originate. In other cases, the cell-growth in the pericardium is accompanied by a free exudation. This always contains fibrin, but in variable quantity, and we are not warranted in attributing this variation to differences in the crasis of the blood; indeed, accumulation of fibrin in the blood must be regarded rather as a consecutive and not as a primary alteration of its composition.

Pericarditis may be caused, although rarely, by injuries, penetrating wounds, blows, concussions, etc.; to this class of cases we naturally annex those in which inflammation has spread to the pericardium from a neighboring organ. It is extraordinarily rare for this malady to attack previously healthy persons as an independent isolated disease. When it does occur, it is chiefly at times when pneumonia, pleurisy, croup, and other inflammatory complaints are rife, and epidemic influence prevails. In such cases it is customary to assume that cold has been allowed to act upon the organism, although this is generally difficult to prove.

Pericarditis occurs much more frequently, allied to other acute or chronic diseases. The most important of these is acute articular rheumatism, particularly when several joints are successively affected. According to Bamberger's carefully-collected statistics, about thirty per cent. of the cases observed have been complicated with acute rheumatism.

Next in frequence, pericarditis complicates Bright's disease, tuberculosis, particularly tuberculosis of the lungs, chronic disease of the heart, and aneurism of the aorta. In all these cases, as it seems to me, the primary disease gives rise to a predisposition to pericarditis; but the latter is not a sequel, only a complication of the primitive complaint, and is not to be regarded as secondary in the narrow sense of the word.

It is otherwise in the pericarditis which attends septicæmia and kindred conditions, puerperal fever, severe scarlatina, small-pox, etc. Here the disease belongs to the consequences, and is not a complication; the infection manifests itself by a series of inflammatory disturbances, attacking the skin, the joints, and the pericardium.

Anatomical Appearances.—In the bodies of many, and especially of old persons, we find upon the visceral surface of the pericardium a number of fine papillæ, consisting of delicate, vascular connective tissue. Still more frequently we find irregular, whitish, flat tendinous deposits, called *maculæ albidæ, lactæ* (*Sehnenflecke*). These, likewise, consist of new connective tissue, springing directly from that of the pericardium, from which they can only be detached by force, and by whose epithelium they are covered. If the pericardial proliferation be of inflammatory origin (a matter still *sub judice*), like the thickening and adhesions of the pleuræ, they appear to proceed from inflammation, which produces a merely nutritive exudation, a pericarditis sicca.

As the growth of villi and the formation of maculæ albidæ are not recognizable during life, but are mere accidental *post-mortem* discoveries, we shall give them no further attention.

In discussion of the subject of exudative pericarditis, our attention will be occupied, first, with the changes undergone by the pericardium; second, the quality and quantity of the exudation.

At the commencement of the disease the pericardium appears more or less reddened, in consequence of a dense capillary injection springing from the deeper parts, with here and there extravasations in the form of irregular, dark-colored, homogeneous red spots. The tissue is relaxed by serous infiltration, and can be very readily torn; the surface, the epithelium having fallen, is dull and void of glitter. The membrane soon takes on a shaggy appearance; fine villi, papillæ, and folds develop by the proliferation and generation of young connective tissue-cells, constituting the first step in the formation of pseudo-membranes, and of the adhesions of the pericardium, which remain after pericarditis.

Pericardial effusions present all the modifications which we have described as occurring in pleurisy. The exudation soon separates into a liquid and a solid portion. The former may be very scanty in quantity, or may amount to several pounds. Small accumulations of it form in the upper and anterior part of the sac, at the root of the great vessels, while the heart gravitates to the lower portion. When in larger amount, the entire sac is distended, the lungs compressed, particularly the lower lobes of the left lung; even dilatation of the thorax, in the region of the heart, may be the consequence.

Although the exudation always contains some young cells or pus-corpuscles, their quantity is often extremely small, and the liquid is then tolerably clear, and either colorless or of a yellowish tinge. If more or less of coagulated fibrin be found in the liquid, it is called sero-fibrinous. A smaller quantity of fibrin imparts a slightly-flaky opacity to the liquid part of the exudation, or may produce a slimy, turbid deposit upon the pericardium. Sometimes delicate fibres, like cobwebs, pass across from one surface to another. This we find, chiefly, in cases in which inflammation has been transmitted from some neighboring organ to the pericardium.

In other cases, the exudation is very heavily charged with fibrin, which is extensively precipitated upon the walls of the pericardium, forming reticulated and villous masses. The surface of the heart acquires the aspect of a cut sponge, or of one of two surfaces smeared with butter, which have been quickly pulled asunder after having been brought into contact. A heart upon which this sort of villous, ragged precipitate has formed, is called *cor villosum* or *hirsutum*. This is the kind of exudation most commonly met with in the pericarditis of acute articular rheumatism.

In many cases an escape of blood from the ruptured capillaries accompanies the exudation, thus producing a *hæmorrhagic exudation*. If there be but little blood mixed with it, the serum has a reddish color; if the flow of blood be considerable, the effused mass may resemble a pure extravasation, and assume a blackish hue. Even the fibrinous deposit, otherwise whitish yellow in color, is stained, either dark or bright-red, by admixture of the blood. Hæmorrhagic exudation sometimes occurs in recent pericarditis, which has attacked cachectic subjects, topers, tuberculous persons, or those suffering from advanced *Bright's* disease. It is still more frequently observed when the inflammation, instead of invading the true pericardium, has attacked the young connective tissue which has developed upon it, and in which very large but delicate and thin-walled vessels form, which are very liable to rupture. In these cases we often find miliary tubercles developed in the young adventitious membrane, besides the hæmorrhagic exudation; and this is what we commonly find in the form known as chronic pericarditis, a disease which, during life, permits of our recognizing repeated outbreaks of the inflammation.

If young cells (pus-corpuscles) be commingled in any great amount with the exudation, the effused liquid becomes yellow and opaque, like thin pus. The fibrinous deposits are remarkably yellow, unelastic, rotten, and even pasty. This we call purulent exudation (*pyopericardium*). It arises precisely like empyema, sometimes from protracted pericarditis, with sero-fibrinous exudation; sometimes the inflammation shows strong tendency to formation of pus-cells from the outset, so that even the recent exudation is purulent. Such a disposition is often seen in the pericarditis of septicæmia, puerperal fever, etc. In pyopericardium, pus-cells sometimes form in the substance of the serous membrane, producing ulceration; although this is more rare than in empyema.

In cases of extraordinary rarity, pericardial effusion becomes putrid, fetid, discolored, emits gas; and here, too, erosion may take place in the membranes. *Ichorous exudation* is the product of such decomposition.

In recent cases, the substance of the heart often suffers no material alteration. In cases of longer standing, however, or when the disease has been very intense, it appears soon to become sodden with serum, softened, and flabby, so that extensive dilatation of the heart supervenes upon the pericarditis. In cases of hæmorrhagic and purulent exudation, the muscles of the heart become very much discolored, flabby, and softened, the epicardial surface undergoing fatty degeneration (*Virchow*). Myocarditis, too, is a not unfrequent accompaniment of the disease.

The effects of pericarditis depend greatly upon the degree of thickening of the pericardium, and the quantity of coagulated matter contained in the effusion. If the thickening be slight, and the amount of fibrin in the effusion small, it is soon absorbed, the liquid first, and then the solids, undergoing fatty degeneration, and thus becoming capable of absorption. Thickening of the pericardium leaves behind it thick tendinous spots, or else adhesions from between the two surfaces, a circumstance of but little moment if the pericardium be but moderately thickened, so that such a termination of the disease may be regarded as a recovery. If the pericarditis be of long standing, the thickening generally becomes so great that permanent and serious disorder remains, even after the exudation has been absorbed. The young connective tissue is converted into a firm fibrous mass, so that the epicardium at last forms a dense indurated capsule around the heart. The parietal surface is usually less thickened, and here, too, if the effusion be fully absorbed, it may be firmly joined to the visceral portion. Frequently, absorption is incomplete; the folds of the pericardium are then only partially adherent; in other places, the residua of the exudation appear in the form of puruloid or cheesy masses, which afterward not unfrequently are converted into a chalky paste, which may seem embedded or impacted in the flesh.

When death occurs at the height of acute pericarditis, or in the course of the chronic form, we find the traces of cyanosis, and not uncommonly discover dropsical effusions in the body.

SYMPTOMS AND COURSE.—As pericarditis hardly ever attacks a person in good health, or appears as a solitary and independent disease, it is difficult to describe its course distinctly. Moreover, when this malady sets in upon some preëxisting disorder, its symptoms often modify those of the latter so little that they are exceedingly apt to be overlooked. When pleuritis or pneumonia extends into the pericardium, a diagnosis, or even a suspicion, of the complication is often impossible without physical examination; and, as the latter too often fails us here, "a participation of the pericardium in the inflammation" often remains undiscovered until the autopsy is made.

When acute articular rheumatism is the complicating disease, it is quite rare for attention to be called to the existence of pericarditis by any rigor, aggravation of the fever, acceleration or retardation of the pulse, pain in the region of the heart, palpitation, dyspnœa, or terror. It should be our invariable rule daily to auscult the chest of a rheumatic patient, even though he do not complain, for all the above-named symptoms may be wanting, and yet pericarditis, and even a copious effusion, may exist. When subjective symptoms do occur, however, pain and palpitation are the more frequent of the signs. The pain usually affects the left side of the epigastrium, and spreads more or less over the chest. It is sometimes piercing, sometimes duller, and is almost always aggravated by a firm pressure upward upon the epigastrium. Excessive pain almost always signifies implication of the pleura or lung in the inflammation. Palpitation is generally met with where the action of the heart is embarrassed, and where the organ has difficulty in fulfilling its task. It is easy to understand that pericarditis can impede the function of the heart through pressure upon it by the exudation, by serous infiltration of its muscles, and by participation of the latter in the inflammation. On the other hand, it is singular that palpitation, and other symptoms indicative of embarrassment of the heart's action, should not be a more common source of complaint. Sometimes the pulse becomes very frequent when the disease sets in; in other cases, it is temporarily retarded. We have already spoken of this latter symptom while upon the subject of endocarditis, and have there expressed our view that it is a matter of pure theory to ascribe these phenomena to irritation of the cardiac ganglia. If the pulse be both frequent and small, pericarditis may assume a strong resemblance to typhus and other asthenic fevers. The sick man is collapsed, is extremely restless, sleeps badly, and starts from his sleep; he becomes delirious, until at last somnolence sets in. The more imperfect and hurried the action of the heart becomes, so much the more marked are the symptoms of circulatory obstruction; the countenance becomes congested and cyanotic, and the breathing rapid. If a fresh obstacle to respiration be added to this passive hyperæmia of the lung, should the lung be compressed by a huge pericardial effusion, the dyspnœa may become intense. The patient lies upon the left side, as it is the left lung which is the most compressed, and freer play is thus afforded to the right side of the thorax, or else he sits upright, or bent forward in bed. Even when the function of the heart is not suffering materially from the effects of the pericarditis, dyspnœa, and very severe dyspnœa, too, may arise through compression of the lung, so that, as acceleration of pulse is not a very common symptom, *pain in the cardiac region, palpitation, and subsequent dyspnœa*, must be pronounced its most frequent subjective signs, if it produce any functional derangement at all.

If pericarditis be a complication of tuberculosis, Bright's disease, chronic disease of the heart, or aortic aneurism, its invasion is equally as insidious as, if not more so than, when it arises in rheumatism. Without physical examination, its diagnosis would be impossible. After long duration, the malady develops a series of symptoms, which we shall describe as chronic pericarditis. If it set in in the course of grave blood-disease, there are absolutely no subjective symptoms. In such maladies the sensorium is usually much benumbed by the asthenic fever, and the great apathy of the patient renders him insensible to pain and distress far more violent than any arising in pericarditis. It would seem that depression of the cardiac action is most intense in cases of purulent effusion, but, without physical proof, we are unable to decide with certainty whether the acceleration and contraction of the pulse, already rapid and small, be due to the prostration or to the pericarditis.

With regard to its course and termination, the forms of the disease which accompany pneumonia, pleurisy, and acute articular rheumatism generally have a favorable issue; the disease is acute, and ends in complete recovery. If, as often happens, it have not given rise to any subjective symptoms, the change for the better is only to be recognized by physical examination. Palpitation, pain, and dyspnœa, if present, usually soon subside, as also does any frequence of the pulse which may appear. This favorable result is far less common in the forms of the disorder which complicate Bright's disease, disease of the heart, tuberculosis, and, rarest of all, in the purulent pericarditis accompanying septicæmia, etc.

Death is not a common consequence of acute pericarditis; that is to say, the disease is not often the sole and immediate cause of death. When it occurs in a rheumatic case, the disordered action of the heart suddenly, or else gradually, increases to cardiac palsy; the pulse becomes small and irregular; the consciousness is completely lost; engorgement of the pulmonary veins produces œdema of the lungs, and the patient dies. Death may be all the more speedy, if the pericarditis be complicated by pleuritis or pneumonia. The termination of tuberculosis, Bright's disease, etc., may also be accelerated by such a complication, but the disease then almost always assumes a chronic form. Cases in which, from the beginning, the effusion has been purulent, almost always end fatally; but it is difficult to decide how much of this evil result is due to the local affection, the pericarditis, and how much to the constitutional disorder which it complicates.

As a third mode of termination, acute pericarditis may pass into a chronic state. A small number of cases of chronic pericarditis proceed from the acute rheumatic form of the disease. It is more common, however, as an accompaniment of the cachectic conditions and cardiac disease which we have so often spoken of. The malady, which probably always at first assumes the acute form, does not get entirely well, and sooner or later (just as in many cases of pleurisy), the inflammation breaks out afresh. The exudation is extremely profuse, the dyspnœa severe. After a while the symptoms abate again; but new relapses often follow, and the disease goes on for months. We have said that the substance of the heart becomes extremely soft, relaxed, and discolored; and, accordingly, we often find the pulse very small and irregular, the veins overloaded, and the patient dropsical. The more copious the exudation in the pericardium, so much the more severe not only does the dyspnœa become, but the cyanosis and dropsy. Much of the blood which ought to be in the arteries is crowded into the veins, and cannot gain access to the right heart; for the latter, compressed by exudation, is unable to dilate, as in other cardiac diseases. It is only in very rare instances that chronic pericarditis terminates in complete recovery. Death by œdema of the lungs and slow suffocation is the most frequent ending, and, in almost every case, the disease is attended by sequelæ:

1. First among the sequelæ of pericarditis stands adhesion of heart and pericardium, to be treated of in the next chapter.

2. We have already learned how dilatation of the heart becomes a sequel of this disease (Chap. II.), and that the longer the attack lasts so much the more is this likely to happen.

3. If the substance of the heart be not degenerated, the dilatation turns into hypertrophy, which is usually total, and is to be set down as a not unfrequent sequel.

4. The nutritive state of the organ suffers under the perpetual pressure of the pericardial exudation, and the constant infiltration of its substance, resulting in atrophy and fatty degeneration.

*Physical Signs—Inspection.*—If the thoracic wall be yielding, and the effusion large, inspection often reveals a distinct bulging of the cardiac region. Ossification of the costal cartilages tends to prevent this prominence, which, therefore, is to be found principally in children and youthful persons.

*Palpation* at the outset of the disease often enables us to feel that the beat of the heart is in its proper position, and frequently, too, that the vigor of the beat is increased. When the exudation is more copious, the impulse is usually weaker than normal, unless the heart be hypertrophied or violently excited. Sometimes the beat is quite imperceptible. It may frequently be felt while the patient is standing upright, but is lost as soon as he lies down, as the heart then sinks back into the liquid, and is separated from the thoracic wall. The impulse often is situated too low down, the diaphragm having become depressed by the accumulation of liquids. *Oppolzer's* statement, that the shifting of the heart-beat as the patient alters his attitude is a characteristic token of pericardial effusion, is incorrect. According to a number of observations of *Gerhardt*, the truth of which I can fully vouch for, the apex of the heart of a healthy person generally moves to the left about two centimetres when he lies upon his left side. Sometimes the hand laid upon the chest perceives a distinct sensation of friction, caused by the rubbing together of the rugged surfaces of the pericardium.

*Percussion.*—If the lung intervene between the pericardium and the thoracic wall, percussion will reveal nothing abnormal even when the exudation is tolerably large (half a pound). At other times an unnatural dulness arises early, which, from the point at which it first becomes perceptible, and the form which it afterward assumes, is one of the most important signs of the disease. At first, as the liquid rises, and the heart takes the deepest position possible, we find a dulness upon percussion at the root of the aorta and pulmonary vessels. It extends upward to the second rib, or even higher, and passes beyond the right edge of the sternum. When very copious, the exudation bathes the entire organ, and the dulness forms a triangle with the base downward, and with an obtuse apex above. The dulness, which always grows broader as it extends lower, often passes far beyond the left mammillary line and the right border of the sternum.

Extension of the dulness to the left, beyond the point at which the apex beats, is a positive sign of the existence of a collection of liquid in the pericardium. *Gerhardt* has pointed out that pericardial effusion forms an important exception to the rule according to which cardiac dulness remains the same, whether the attitude be erect or recumbent, as in the latter case its limits become from one-third to one-half larger.

Upon auscultation, unless the heart be hypertrophied, or in violent action, its sounds are remarkably feeble, and often nearly inaudible. The disproportion between the extensive dulness and the feeble impulse and lowness of the heart-sounds is an important indication of pericardial effusion. In addition to this, there are, in most cases, friction-sounds which suggest the idea of scraping, rubbing, and scratching. These friction-sounds are unlike those of pleuritis, which are only audible before the pleural surfaces are separated by the liquid, or after the liquid part of the effusion has been absorbed, as they sometimes may be heard when there is a great deal of water in the pericardial sac. As the sounds are produced both by the rubbing up and down of the heart against the thoracic wall and its rotation upon its long axis, after the opposing surfaces have lost their primitive smoothness, and as the movements of the heart are of far longer duration than the normal sounds which it causes, these friction-sounds, although rhythmical, are hardly ever isochronic with the normal cardiac tones, but outlast them, making a prolongation, or sometimes preceding them.

About the lower lobe of the left lung the percussion-sound is often flat, from pressure, and we must beware of mistaking it for pleuritis. The presence of pectoral fremitus will guard against error.

Diagnosis.—Pericarditis is most apt to be mistaken for endocarditis. The functional disturbances, when they occur, are very much alike in the two diseases, although pain about the heart is far more common in pericarditis, as is also the case with dyspnœa and the cyanosis. As it often happens, however, that neither of them furnishes any subjective symptoms whatever, differential diagnosis must mainly depend upon physical exploration: 1. In the first place, in endocarditis, we never find prominence of the cardiac region, which, although not common, does sometimes appear in pericarditis. 2. The form of the tract of abnormal dulness affords an important clew. In endocarditis the dulness may become abnormally widened in a few days, as when dilatation of the right ventricle occurs early. In pericarditis the dulness almost always begins in the vicinity of the great vessels, and afterward assumes a triangular form. If the left border of the dulness reach beyond the apex, the right considerably surpassing the right edge of the sternum, effusion is present in the sac. We have already dwelt upon the significant fact that, notwithstanding the extent of the dulness, the heart-tones are low, and the beat feeble and inaudible when the patient lies down. If the dulness commence at the second rib, we must take notice whether or not the diaphragm and heart be not pushed upward. If so, it will be impossible to form any positive conclusion as to the presence of liquid in the pericardium. The possibility of the existence of aneurism of the aorta, of excessive dilatation of the right auricle, of infiltration of the anterior edges of the lung, and of retraction of the lung, which allows a larger portion of the pericardium to come into contact with the thoracic wall, must also be excluded ere the diagnosis of pericardial effusion can be regarded as established. Sometimes, notwithstanding the existence of a very large effusion, the cardiac dulness is not increased, although in the vicinity of the area of dulness the percussion-sound is somewhat flat. In these cases the anterior edge of the lung has become immovable, owing to adhesion of the pulmonary and costal pleuræ.

3. The murmurs heard in the heart usually permit of a conclusion as to the nature of the existing disease. In the first place, their quality affords some information. Not only the pericardial sounds, but many of those arising in the heart are friction-sounds. In the one case, the roughened surfaces of the pericardium rub together; in the other, the roughened endocardial surface is rubbed by the current of the blood; but, in many instances, the sounds are so distinctly those of scraping or scratching, that we can have no doubt but that they proceed from the pericardium. The points at which they are best audible is of more importance. As it is mainly the right side of the heart which lies in contact with the side of the chest, and rubs against it during its diastole and systole, pericardial sounds are very often heard over the right ventricle, where endocarditis and valvular disease are very rare. The time at which the sounds are heard is of great moment. In endocarditis, they are isochronic with the heart-sounds, and supplant them. In pericarditis they precede the normal sounds, or come after them. When the beat of the heart is very rapid, it is hard to say if the false sounds be isochronic with the normal ones or not.

The extension of the sounds exhibits a further difference (*Bamberger*). In pericarditis they are sometimes confined to a very small spot; in endocarditis they are transmitted along the current of the blood. Lastly, as the heart rises and falls in the liquid around it, pericardial sounds are much more liable to change with alteration of attitude in the patient than the endocardial murmurs.

Rhythmical friction-sound of the pleura may arise in consequence of inflammation of that portion of the pleura which overlies the pericardium, the roughened costal pleura being made to rub against the pulmonary pleura by the beating of the heart. This extra-pericardial friction can only be distinguished from the intra-pericardial sounds when it ceases entirely during inspiration. I have seen one very well-marked case, in which it could be demonstrated, by means of auscultation and percussion, that the expanded lung entered the mediastino-costal sinus during inspiration, and separated the roughened surfaces of the pericardium and costal pleura.

It is not always easy to determine the character of the exudation in the cases in question, although the cause of the disease and its duration may enable us to form an opinion. The pericarditis which complicates rheumatism is, if recent, almost always accompanied by a sero-fibrinous effusion. That of septicæmia is nearly always purulent; the chronic variety often has a hæmorrhagic exudation. It would be unsafe to infer the nature of the effusion from the character of the constitutional disorder, as the latter depends more upon the primitive disease than upon the form of exudation. Even physical research only informs us, by means of friction-sounds, of the presence of rugged layers of fibrin. When the exudation is purulent, the surfaces are not rough enough to give rise to friction sounds.

Prognosis.—As we have already said, pericarditis, supervening upon rheumatism, very rarely causes death, and this is also the case with primary idiopathic and traumatic forms of the disease. Out of twenty cases, seventeen of which were rheumatic, *Bamberger* did not find one fatal case. The prognosis is favorable also where the malady proceeds from pneumonia or pleuritis, as is shown by *Bamberger's* statistics. It is quite otherwise where it complicates incurable disease, as it then nearly always hastens, if it does not actually bring about, a fatal termination.

In discussing the terminations of pericarditis, we have seen how great the number of sequelæ is, by which it is liable to be succeeded. According to their nature, these exert more or less influence upon after-life.

Treatment.—Upon the subject of treatment of pericarditis, we may refer in great part to what we have already said regarding pleuritis and endocarditis.

General blood-letting is never required in pericarditis as such. Its employment is to be confined to the very few cases in which the repressed outflow from the veins into the heart causes symptoms of pressure upon the brain, and demands a reduction of volume in the circulation. Local blood-letting moderates the pain somewhat, and is indicated where it is troublesome. It is best to apply from ten to twenty leeches, according to circumstances, to the left edge of the sternum. The effect is astonishing in most cases. The use of cold deserves great reliance. Even ice-bladders have been applied upon the cardiac region. Digitalis is suitable in cases where the beat of the heart is very frequent and insufficient, causing cyanotic and dropsical symptoms. Its effect here is often very marked. Calomel and blue ointment, in spite of the praise of English physicians, are not only useless but hurtful. As to the employment of diuretics, drastics, preparations of iodine, and blisters, what we have said, while treating of pleuritis, applies equally well here. Impoverishment of the blood, which occurs in protracted cases, requires nourishing diet and iron. Threatening heart-palsy demands stimulus.

When a recent pericarditis comes on in acute rheumatism, we may assume that it will do as well, and perhaps better, without treatment. As long, therefore, as nothing save the physical signs betrays its existence, it is better to refrain from active interference. The astonishing number of recoveries in *Bamberger's* collection of cases occurred under a thoroughly expectant treatment. It is only under conditions mentioned above that we should apply leeches, cold, etc. In order to promote absorption, *Bamberger* lays stress upon the application of warmth and moisture, and especially upon flying blisters. Paracentesis is to be performed when the distress of the patient, especially from the dyspnœa, imperatively demands aid. The result is merely palliative, as a rule; but, even to afford the sufferer opportunity, after the operation, to pass the night in his bed (perhaps for the first time in a long period) and to enable him to sleep a little, is a great gain. Whether in other cases the operation can effect a radical cure, our limited experience does not permit us to decide. Particulars of the operation are to be found in the hand-books on surgery.

## CHAPTER II.

### ADHESION OF THE HEART AND PERICARDIUM.

ANATOMICAL APPEARANCES.—Adhesion of the pericardium and heart is one of the consequences of pericarditis. Its pathogeny and etiology have been given in the previous chapter. The adhesion is sometimes partial, sometimes total. Sometimes it consists in a firm agglutination of the surfaces, sometimes long bands and fibres are the media of connection. In a clinical point of view, the condition of the epicardium is of much more importance. There is occasionally so little thickening of the adherent pericardial faces that the pericardium seems to have disappeared; in other cases the epicardium is converted into an indurated, unyielding case, in which we find masses of even a bony hardness. Again, in circumscribed spots where the fusion of the two surfaces is incomplete, remnants of effusion now and then exist, as we have already described.

Symptoms and Course.—As but a small portion of the pericardium is attached to the thoracic wall, and even that is held by loose cellular tissue, a simple adhesion of the two surfaces does not seem materially to interfere with the movements of the heart. Functional disturbances, observed to accompany this condition, usually depend upon a concomitant degeneration of the heart, valvular disease, or, perhaps, upon a former carditis. The effect is very different when the organ is enclosed within and adherent to a dense fibrous case, often of the consistence of cartilage. Such a condition reduces the propulsive power of the heart in the very highest degree. The pulse becomes extremely small and almost always is very irregular. Dyspnœa, cyanosis, and dropsy appear all the earlier, as the substance of the heart is nearly always degenerated. Physical examination must decide to what source disorders of the circulation are due.

A lack of difference between the percussion-sounds during inspiration and those during expiration has been given as one of the physical signs of pericardial adhesion; but, whether heart and pericardium be or be not adherent, the lung will still intervene between the latter and the side of the chest with every deep inspiration, and, conversely, will recede when a forced expiration is made. In this respect, then, the signs will remain unaltered, unless, indeed, the outer surfaces of the pleura and pericardium be grown together (*Cejka*). There is a second symptom, of greater value. Sometimes, at the point whereat we ought to feel the beat of the apex, instead of rising, we see the intercostal space sink with every beat. This phenomenon we explain as follows: The heart is shortened during systole, and a vacuum would form, were not the space filled, either by the descent of the heart or the depression of the intercostal space; but, if heart and pericardium be adherent, no descent can take place, hence depression of the intercostal place must substitute it. This symptom is all the more important, if, during diastole, we find the space rise again, when, upon cessation of the systolic suction, the heart again becomes elongated, and the apex returns to its position. This symptom, too, is often wanting in many cases of pericardial adhesion. If the pleura and pericardium be not adherent, the lungs may occupy the vacancy left by the withdrawal of the apex during systole, and *vice versa*.

On the other hand, a systolic depression of the region over the apex may depend upon other causes than that of adhesion of the heart and pericardium. If the latter be likewise attached to the spinal column, the lower half of the sternum will also be drawn down by the systole of the ventricle. Moreover, according to *Friedreich*, in such cases there sometimes is a peculiarity observable about the veins of the neck. We see, namely, when the sternum, after having been drawn down by the systolic movement, springs back again with the diastole, thereby creating an expansion of the chest, that the veins collapse. In *Friedreich's* case this phenomenon only lasted for a limited time, and ceased as the action of the heart, and with it the systolic depression, grew more feeble. Thus, in solitary cases, physical examination may inform us of the existence of pericardial adhesion. In the majority of instances, however, the statement of *Skoda*, in the first edition of his book, still holds good, that "no symptoms are discoverable, through percussion and auscultation, which can be ascribed to adhesion of the heart and pericardia."

## CHAPTER III.

### HYDROPERICARDIUM.

ETIOLOGY.—Hydropericardium depends upon an increase of the normal liquor pericardii, a transudation which contains but little albumen. We have already seen how a decrease in the size of the heart, by reducing the pressure upon the pericardium from within, results in an increase in quantity of the liquid in the sac. The same thing takes place when the lungs become adherent to the pericardium and are reduced in volume, either from atrophy, failure to regain their normal size after absorption of a pleuritic effusion, or contraction from chronic pneumonia. This form of hydropericardium is analogous to the increase in the amount of cerebro-spinal liquid which takes place in atrophy of the brain, and, as the latter is called hydrocephalus ex vacuo, so hydropericardium ex vacuo would be a suitable name for the former.

A second form of hydropericardium is that which arises from an obstruction of the veins of the right heart. An abnormal pressure is thus thrown upon the pericardial veins, and dropsy of the sac results just as it takes place in other serous sacs, or in the subcutaneous cellular tissue. The collections of water in the pericardium arising in valvular disease of the mitral, emphysema, cirrhosis of the lungs, and in other diseases in which the right side of the heart is overloaded, all belong under this head. In all these cases dropsy may be of earlier occurrence in the pericardium than in any other part of the body.

It is otherwise in the third form, in which dropsy of the pericardium, like that of other organs and structures, is to be regarded as the effect of a "dropsical crasis." Appearing in diseases in which the blood has a tendency to lose its albumen, and for its serum to transude, as in Bright's disease, chronic affections of the spleen, cancerous cachexia, etc., the pericardium is not usually affected until a late period.

ANATOMICAL APPEARANCES.—According to the explanation of the foregoing paragraph, only collections in the pericardium, of a liquid containing but little albumen, are to be regarded as hydropericardium. If the liquid contain fibrin, it belongs to the inflammatory effusions. Sometimes small quantities of disintegrated blood are mingled with the serum. In such cases the nutritive state of the capillary walls has deteriorated so that they become ruptured. The frequent occurrence of small hæmorrhages into the skin (*petechiæ*), in general dropsy, is an analogous condition.*

The quantity of the liquid effused is very variable. A collection of an ounce or an ounce and a half of liquid in the sac is not to be regarded as pathological. In cases which are not rare, the amount may be as much as four or six ounces; in others, particularly when the affection arises from disturbance of the circulation, it may exceed several pounds. When the effusion is very large, the pericardium is dull-white and lustreless, the fat has disappeared from about the heart. Sometimes its connective tissue is œdematous.

Copious dropsical effusion into the pericardium distends it, compresses the lungs, and dilates the thorax exactly like pericardial exudations.

SYMPTOMS AND COURSE.—Our remarks upon the subject of hydrothorax are equally applicable to that of hydropericardium. Although, to the minds of the ancient physicians, "water on the heart" used to be a most formidable malady, as even now is the case among the laity, yet it has no real title to rank as an independent disease. But not only is accumulation of liquid in the pericardium always a secondary affection, depending either upon some derangement of the circulatory or respiratory apparatus, or else upon a morbid condition of the blood, but the very symptoms imputed to water on the chest, and so much dreaded, proceed chiefly from the primary disease, and are not caused by the pericardial effusion. When, prior to the introduction of physical examination, a diagnosis of hydropericardium was often confirmed *post mortem*, it was due to the fact that the symptoms upon which the diagnosis was based nearly always arose from such diseases as emphysema and valvular disease of the heart, which ultimately resulted in dropsical affections, and therefore in effusion into the pericardium.

A large serous effusion into the pericardium undoubtedly has the effect of aggravating the dyspnœa arising from the primary disease. Such an effusion often helps to prevent the patient from lying down without danger of suffocation, and compels him to sit day and night leaning forward upon his chair or bed. Moreover, the pressure exerted by the liquid upon the heart and mouths of the great vessels impedes the systemic circulation, causing the jugular veins to swell up, and aggravating the cyanosis and dropsy. Frequent as is the coexistence of such symptoms with hydrops pericardii, yet the presence of every one of them does not afford sufficient ground for a positive diagnosis of hydropericardium, unless supported by the evidence drawn from physical exploration. All these symptoms may be present without there being any increase in the amount of the pericardial liquid. Upon physical examination, the prominence of the region of the heart is observable, although in a less degree than in cases of inflammatory effusion. The depression of the intercostal spaces is not obliterated. The impulse of the heart is very feeble, and is often quite imperceptible, especially when the patient lies upon his back. When the effusion is large, and provided that the lungs are capable of retraction, the cardiac dulness is extended, and has the same shape and exhibits the same modifications, upon change from the upright to the recumbent attitude, which already have been described as characteristic of pericardial effusion. It happens more frequently in this affection than it does in pericarditis, that the lung is unable to retract, owing either to emphysema or to adhesions of the costal and pulmonary pleuræ. In such cases, notwithstanding the existence of a very large effusion, the area of dulness is not extended. Upon auscultation, unless the valves of the heart be diseased, the heart-sounds are pure though feeble. Friction-sounds are never heard.

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* We shall treat of fibrinous dropsy in cancer of the pericardium, just as we have spoken of it in cancer of the pleura.

TREATMENT.—All the measures recommended for the treatment of hydrothorax are equally applicable to that of hydropericardium. The only rational procedure is to treat the primary disease. It rarely is practicable to reduce the liquid in the pericardium by means of diuretics and drastics.

## CHAPTER IV.

### PNEUMOPERICARDIUM.

AIR sometimes enters the pericardium through a perforating wound of the thorax; in other cases the pericardial sac suffers perforation by some destructive morbid process, and air is admitted into it from some organ which naturally contains air. I have observed one instance of this kind (which has been reported by *Dr. Tütel*, my assistant at the time, in the German Clinic), in which pneumopericardium arose after perforation of the pericardium by carcinoma of the œsophagus. Other cases have been reported of perforation of the pericardium by ulcers of the stomach, cancer of the stomach, or superficial cavities in the lungs. Finally, gas sometimes is generated in the pericardium, by the decomposition of the effusion which it contains.

Upon *post-mortem* examination, the pericardium is usually much distended, partly by air and partly by a purulent or sanious liquid. The latter is the product either of a recent pericarditis, caused by the entrance of air, or cancerous discharge, or of broken-down pulmonary tissue, into the pericardial sac, or else, if the pneumopericardium be the result of a generation of gas from a putrefying exudation, of a pericarditis of long standing. Upon puncture of the distended sac, the air usually escapes with a hissing sound.

Pneumopericardium is far less common than pneumothorax, and nearly always is easy of recognition. It is true, the subjective symptoms, arising from perforation of the sac and of entrance into it of air and *débris* of the tissue, are not very characteristic. Besides, the occurrence is usually attended by severe collapse, in which the patient lies in a state of apathy, making no complaint, and, if questioned, replying with hesitation and incompleteness. Even at some distance from the patient a peculiar, clear, splashing sound can be heard, which comes and goes with short, rhythmical intervening pauses, and which, beyond all question, is caused by the agitation produced in the liquid contents of the pericardium by the movements of the heart. In my case this splashing sound was distinctly audible to the room-mates of the patient, who lay at the other end of the ward. Upon *inspection*, if the thorax still be flexible, the prominence of the cardiac region and the obliteration of the intercostal depressions are very marked. The cardiac impulse is indistinct or imperceptible. Upon *percussion*, there is no cardiac dulness, and, indeed, the percussion-sound about the region of the heart is extremely full, clear, and tympanitic, often having a distinct metallic ring. Upon making the patient sit up, or upon making him bend forward, the beat of the heart becomes somewhat more perceptible, and, as the air now rises and the liquid presses forward, the former clear sound is replaced by a dull one. Upon auscultation, either nothing can be heard excepting the above-named metallic splashing, or else we may also hear feeble heart-sounds and friction-sounds.

With exception of cases of traumatic origin, this disease, as a rule, rapidly proves fatal. The collapse and severe pericarditis which almost always accompany pneumopericarditis sufficiently account for this. Recovery from traumatic pneumopericardium has been observed repeatedly. Of course, the treatment of this affection can only be a treatment of symptoms, and in most cases it is limited to an exhibition of stimulants.

## CHAPTER V.

### TUBERCULOSIS OF THE PERICARDIUM.

TUBERCLES in the tissue of the pericardium are only seen in acute miliary tuberculosis. The grayish nodules here visible do not undergo further metamorphosis, and the patient dies, consumed by fever, without betraying any symptoms of the existence of tubercles in the pericardium.

It is far more common for tubercles to form in the young pseudomembranes which develop on the pericardium in the course of a chronic pericarditis. We nearly always find a hæmorrhagic effusion in the sac in these cases, and observe its walls to be studded with drusy prominences, which are at first translucent, but may afterward become yellow and cheesy, although they rarely soften into "tubercular pus." The symptoms of this form of tubercle of the pericardium are undistinguishable from those of chronic pericarditis.

## CHAPTER VI.

### CANCER OF THE PERICARDIUM.

CANCER of the pericardium is almost always an extension of cancerous disease from the sternum or mediastinum. Sometimes it grows diffusely, so that a large part of the sac degenerates into cancer; sometimes it forms solitary rounded masses, or flat nodules upon the membrane. More rarely it appears independently after extirpation of an external cancer; and then other nodules upon other organs and upon other serous membranes are nearly always found. The formation of cancer in the pericardium is nearly always combined with a collection of liquid within the sac, which, like the liquid found in cancer of the peritonæum or pleura, contains "fibrin of tardy coagulation." It is possible, only in the very rarest instances after removal of a cancer of the breast, to diagnosticate the formation of cancer of the pericardium by the evidence of a gradually increasing effusion in the sac.

# SECTION III.

## *DISEASES OF THE GREAT VESSELS.*

### CHAPTER I.

### INFLAMMATION OF THE COATS OF THE AORTA.

ETIOLOGY.—Inflammation of each of the three tissues of the aorta, the adventitia, the media, and the intima, is best studied by itself, just as we have already successively discussed pericarditis, myocarditis, and endocarditis.

Acute inflammation of the tunica adventitia is rare, and hardly ever occurs, excepting when inflammation or ulceration of the lymphatic glands, the œsophagus, the trachea, or other neighboring organ, extends into the aorta. Chronic inflammation is far more common; but neither is it primary, being allied almost always to pericarditis and attacking the root of the aorta; or else to endarteritis, when its action may be very extensively diffused.

The tunica media often takes part in inflammation of the adventitia. In chronic inflammation of the intima, too, the media is almost always diseased, but is not often inflamed. It is much more commonly the seat of simple atrophy or of fatty degeneration.

From *Virchow's* point of view, chronic inflammation of the internal coat of the arteries is to be regarded as one of the most frequent of diseases. The reason for classing the gelatinous and semicartilaginous thickening of the inner arterial tunic (see below), which forms the incipient stage of ossification and atheroma of the arterial walls, among the parenchymatous inflammations, is due to the fact that, in this disease, we undoubtedly have to do with an active process, with generation of cells, and that in many cases it can be shown that these nutritive disturbances owe their origin to certain irritants which have acted upon the tunics; as, undue strain, or distention (see pathogeny of endocarditis). In other cases, indeed, it cannot be proved that the arteries have been subjected to special irritants; as, however, the anatomical changes are precisely the same, we may assume that the sources of irritation exist, but have escaped our observation.

Endarteriitis deformans, as we may call chronic inflammation of the intima, according to *Virchow*, is an extremely common disease of advanced age; and it is always at the points most exposed to strain or distention, such as the ascending portion and arch of the aorta and the places of origin of the vessels which pass off laterally, that the disease is most apt to occur. In the second place, the malady is most frequently found to affect gouty, rheumatic, or syphilitic persons, as well as drunkards. We are not at liberty, however, to go so far as to suppose that in these cachectic subjects the disease proceeds from the composition of the blood, that an irritant circulates in the latter which excites the internal coat of the artery to the point of inflammation.

Finally, endarteriitis accompanies hypertrophy of the heart in young subjects who are not cachectic, and here it seems to attack by preference dilated portions of the arteries. These cases furnish strong evidence of dependence of the disease upon local injury to the vessels.

ANATOMICAL APPEARANCES.—We rarely have opportunity to see purulent and ichorous collections in the tissue of the adventitia. Indurated thickening of the cellular tissue, as a residue of chronic inflammation, is a far more common discovery. At first the calibre of the artery usually is narrowed; afterward it generally becomes widened.

Inflammation of the tunica media begins with a speckled redness, which has its seat beneath the internal coat. The spots soon become of a whitish or yellow color, are elevated above the inner surface, and resemble small pustules. At first a mere sprinkling of the infiltration, in the form of amorphous granules, takes place upon the tissue, which still remains firm. It afterward liquefies, and pus forms, so that actual abscesses are established in the wall of the artery.

Chronic endarteriitis commences with relaxation and infiltration of the tunica intima. Two forms of it occur, distinguishable according to the grade of infiltration, and which have often been described as different stages of the same disease. In the first form, that of gelatinous thickening of the inner coat, a gelatinous, moist, pale-reddish layer seems to lie upon the inner surface of the artery, sometimes in circumscribed spots, sometimes spread over a wider surface. These apparent deposits readily admit of being crushed or displaced in the form of jelly. They consist mainly of a liquid resembling mucus, in which fine elastic fibres and round or spindle-shaped cells lie embedded. They are immediately connected with the tunica intima, and are covered by its epithelium.

In the second form, that of semi-cartilaginous induration, we find opaque, bluish-white plates, like boiled white of egg, lying upon the inner surface of the artery. Here, too, the tissue of the tunica intima is softened and infiltrated, but it remains firmer and more consistent than in the other form, and it afterward assumes a cartilaginous hardness. Under the microscope, numerous fusiform and reticulate cells can be seen in the semi-cartilaginous variety, but, above all, broad fasciculi of connective tissue are visible, which plainly form an immediate continuation of the lamellar fibres of the tunica intima.

The further changes which these inflammatory products undergo are: 1. Fatty metamorphosis; 2. Calcification or ossification.

In the gelatinous thickening, fatty metamorphosis begins chiefly in the superficial portion, commencing in the cells of which we have spoken, while the intermediate substance breaks down, and the surface becomes rough and tufted. This process is called "*fettige Usur*"—"fatty consumption."

In semi-cartilaginous thickening, fatty metamorphosis begins in the deeper layers. Here, too, at first, numerous drops of fat are deposited around the nuclei of the connective tissue-cells, so that they become transformed into star-shaped cells of fatty granules. The cell membrane ultimately perishes, and the fat molecules are liberated. The bundles of connective tissue also break down, and thus, deep in the interior, a pea-soup colored, fatty paste is formed, consisting of fat molecules, numerous crystals of cholesterin, and *débris* of connective tissue, constituting *true atheroma.* As long as the greasy paste remains separated from the current of the blood by a thin film of the internal coat, it is called an atheromatous pustule. Afterward, when the covering has broken down, after its contents have been washed away, and an irregular loss of substance, with ragged edges, has formed, we speak of an atheromatous ulceration. Atheroma and "usur" bear the same relation to one another as abscess and ulcer.

Calcification depends upon a deposit of salts of lime in the deeper layers of the semi-cartilaginous thickening. In the plates of lime thus formed we sometimes find bodies analogous to bone-corpuscles, jagged in form, and furnished with prolongations, which are the residua of connective tissue-cells, so that we then are warranted in employing the term ossification. While the smaller arteries may become converted into tubes, with rigid walls, from deposit of earthy matter, ossification of the aorta usually appears in the shape of separate plates and scales, of variable size, which form shallow depressions upon the inner surface of the vessel, and which are separated from the blood by the intervention of a thin film of the tunica intima. By-and-by the ossification reaches the surface itself; the scales of bone are completely exposed, and are sometimes washed loose by the blood, and then form projections, upon which the fibrin of the blood is very apt to precipitate itself.

At the outset of endarteriitis, the tunica media does not become perceptibly altered. In advanced atheroma it grows yellow, relaxed, and fissured. Large deposits of fat form between its lamellæ. The media is generally atrophied, and thinned by ossification of the intima. In the beginning of the process the adventitia is also normal, but afterward becomes swollen, thickened, and indurated.

In many cases we find all the various phases of the disease alongside of one another in the aorta: in one place gelatinous or semi-cartilaginous induration; in another, atheromatous pustules; here again ulceration; there calcification, in a slight depression, covered by the tunica intima; and at some other point we find plates of bone projecting free into the aorta.

SYMPTOMS AND COURSE.—No clinical description of acute inflammation and ulceration of the adventitia can be given, as, in the few instances in which the process has been watched, the disease has always been complicated by other grave disorders. This is also the case in chronic inflammation of the adventitia, and in the instances of abscesses observed now and then in the tunica media.

Chronic inflammation of the tunica intima and its results, generally comprehended under the term atheroma, in its wider sense, furnish but few symptoms as long as they do not cause aneurism, rupture, or stoppage of one of the smaller arteries by detachment of a clot. We shall consider the subjects of aneurism and rupture in the second and third chapters. The results of embolism, as far as they affect internal organs, are treated of in various chapters of this work.

If, in consequence of degeneration of its coats, the aorta have lost its elasticity, and if, too, its branches take part in the disease, the demands upon the heart are increased, and hypertrophy arises (see hypertrophy of heart). If the chronic inflammation spread from the arterial wall to the valves, insufficience and stenosis may be the result. Hypertrophy often fails to take place, owing to depression of the general nutritive condition, or it is not sufficiently pronounced to compensate for the impediment which degeneration of the aorta and its ramifications presents to the circulation, or else the hypertrophy is soon converted from a genuine to a false hypertrophy, from degeneration of the substance of the heart. Symptoms of retarded circulation and overloading of the venous system then arise, cyanosis, dropsy, and suppression of urine, as described in a previous chapter.

The most important token in the diagnosis of chronic inflammation of the inner coat of the aorta is the evidence of the existence of similar disease in the peripheral arteries, as the inference is thus warranted that the affection is also present in the aorta, in a still more advanced stage of development. As the vessels have become dilated, and their walls more rigid, the pulse generally feels hard and full; the course of the elongated arteries is remarkably sinuous, their curvature increasing with the beat of the pulse, which becomes visible. The artery, even when undistended by the current of the blood, can usually be felt as a hard, irregular cord.

As long as there is no aneurismal dilatation of the aorta, percussion and auscultation do not aid the diagnosis. In rare cases, false murmurs arise, in consequence of roughness of the inner coat of the aorta, and where there is no deformity at the ostia.

According to *Bamberger*, the first sound of the aorta is often dull, muffled, or even inaudible; the second, particularly if the walls of the great vessels be studded with bony plates, and as long as the valves retain their efficiency, has a remarkably loud and metallic ring.

## CHAPTER II.

### ANEURISM OF THE AORTA.

ANEURISMS, produced by wounds, belong to the province of surgery. Spontaneous aneurism, that is to say, partial dilatation of a vessel caused by degeneration of its walls, is the only form of the disease which occurs in the aorta.

Uniform dilatations of the entire tube, such as arise in hypertrophy, and which take place above stricture of the vessel, are not regarded as aneurisms.

ETIOLOGY.—The degeneration of the aortic wall, which most frequently gives rise to aneurism, is the result of the chronic endarteriitis described in the previous chapter, and known as atheroma.

Next in frequence, as a cause of aneurism, is simple fatty degeneration of the inner and middle arterial tunics, a disease which we have purposely avoided mentioning hitherto, as it has nothing in common with the inflammatory affection previously described. In simple fatty metamorphosis there is no preliminary thickening, and cell-growth in the tunica intima; but, from the very outset, we find opaque, whitish or yellowish-white spots, grouped in a peculiar manner, and but slightly prominent above the surface, which consist of deposits of fat molecules in the tissue of the arterial coats.

Thirdly, simple atrophy, and thinning of the aortic wall, which seems to be by no means uncommon among elderly people, may be a cause of aneurism.

Whether palsy of the vaso-motor nerves be also a cause of this disease (*Rokitansky*), is questionable, at all events, as regards the aorta, which is but poorly provided with contractile elements.

In consequence of these changes, particularly in the middle coat, the aorta loses its elasticity, sometimes at a circumscribed spot, sometimes throughout a larger portion of its extent, and gradually yields and becomes dilated by the pressure of the blood. Not unfrequently, however, upon the occasion of some sudden strain, the tunic of circular fibres seems to give way suddenly, and the dilatation of the wall, which now consists only of the adventitia and intima, goes on more rapidly. Many persons suffering from aneurism believe that they know the period, or even the moment, from which their malady dates, assigning, as a cause, some violent muscular effort, the lifting of a heavy burden, etc. It has already been remarked that a general contraction of the muscles, by compressing many of the capillaries, must throw an increased strain upon the aorta. A violent jar of the frame seems to have a similar effect; at least, many patients date their affection from some fall from a great height, or the like. Such accidents will not cause aneurism in a healthy subject; and, in many cases, an acknowl edgment of the immediate causes of the complaint is only forced upon the patient by the examiner.

Aneurisms are rare in young people. They occur chiefly in persons of somewhat advanced age, in whom chronic inflammation of the arterial coats is a very common affection. Men are much more frequently attacked than women; but as the majority of aneurisms are found in persons who habitually make violent muscular efforts, this difference may be accounted for by the difference in the occupations of the sexes.

Anatomical Appearances.—*Scarpa's* classification was mainly based upon the number of coats which could be counted in the wall of an aneurism. If the wall contained all three tunics, it was an *aneurisma verum;* if covered by the adventitia alone, it was an *aneurisma spurium*, or *mixtum externum*. If, again, the wall consisted of a protrusion of the intima through an opening in the media, the pouch being either bare or covered by the adventitia, it was an *aneurisma mixtum internum seu herniosum*. This classification has been abandoned as unpractical. An aneurism may belong to the first class (aneurisma vera) at the period of its commencement, and, as it grows, become an aneurisma spuria; and, indeed, in the same tumor, one half of it may be of the true kind, and the other of the false.

Classification of aneurisms, according to their form, is of more importance. Thus we distinguish the circumscribed and the diffuse aneurism.

A diffuse aneurism involves a considerable portion of the vessel, and its entire calibre. If the dilatation terminate abruptly, it is called a *cylindrical aneurism.* If it decrease gradually, it is a *fusiform aneurism.* Diffuse aneurism is always a true one, in *Scarpa's* sense, and is most commonly met with in the ascending and transverse portion of the arch of the aorta. It is very often combined with the form next to be described, that is to say, circumscribed pouches often form upon the dilated portion of the artery.

Circumscribed aneurism consists in the dilatation of a shorter portion of the artery. Here, too, the artery is sometimes widened in all directions, the tumor involving its entire diameter. Far more frequently, however, one side alone is dilated, and the aneurism, whose walls form an angle with those of the normal part of the vessel, assumes the appearance of a tumor situated on its side. Secondary pouches, in the form of elevations of varying size, are often observed upon these sac-like dilatations, just as in the other kind. At the outset, the disease almost always bears the character of a true aneurism of *Scarpa*, consisting of all three of the aortic coats; but, when the sac has attained some magnitude, the inner tunic only extends for a short distance into it. When at its period of fullest development, the middle tunic, too, dwindles, and finally disappears totally, while there still remain traces here and there of the tunica intima, in a state of degeneration.

Aneurisms attached by a neck must be regarded as a peculiar species of the sac-like form. In these cases, a very small spot on the arterial wall gives way. If the dilatation be large, the wall of the aneurism wraps itself around that of the artery. Thus a duplicature is formed, which, looked at from within, presents a prominent ridge, while, from without, the tumor seems to have been constricted at its base. In these saccular aneurisms, the tunica media can only be traced for a short distance, and soon disappears on the far side of the neck, the wall then consisting of the intima and adventitia (*aneurisma mixtum internum seu herniosum*). When very large, all the tissues gradually disappear under the pressure; and the adjacent structures, to which the aneurism becomes adherent, finally furnish its wall. If the enlargement be slow, the new wall may become very firm by proliferation of the connective tissue; but, if rapid, the wall remains thin, and soon bursts. If the tumor encounter any resisting body, such as bone, the latter undergoes absorption (usur), just like the sac-wall, and, after destruction of the periosteum, the bone is laid bare, and projects naked into the pouch.

In the cavities of aneurisms of large size, especially in the sac-shaped ones, we almost always find deposits of fibrin arranged in separate layers. Those attached to the walls are yellow, dry, and firm;

those next the blood are reddened and soft. Here and there, between their layers, there are frequently deposits of brownish-red or chocolate-colored blood.

The vessels proceeding from the sac, independently of the contraction to which they are liable by ossification, are not unfrequently blocked up by clots of fibrin, and are impervious to blood. In other cases, their mouths are stretched into narrow slits, and in others again they may be narrowed or closed by pressure of the tumor. The condition of the vessels springing from an aneurism is important in a diagnostic point of view.

The size of aortic aneurism varies. Within the pericardium they rarely attain any great magnitude, but soon give way. When they originate beyond the pericardium, however, they may grow to the size of a man's head.

The effect of an aneurism of the aorta upon the parts about it depends upon the amount of displacement and pressure which it inflicts. The trachea, the bronchi, the œsophagus, the great vessels of the thorax, or the nerves, may be dislocated or atrophied by the compression to which they are exposed. Atrophy, or "usur," of the bones may even open the spinal canal, and destruction of the bone and cartilage of the thorax may permit the aneurism to emerge as a prominent tumor, covered only by soft parts.

As partial dilatation of the aorta augments the labor of the heart, that organ is almost always hypertrophied.

Spontaneous cure of an aortic aneurism, by complete solidification of the sac by means of coagula and subsequent atrophy, is one of the greatest of rarities. Other forms of spontaneous cure, which sometimes occur in peripheral aneurism, are impossible in the aorta.

When death does not result from the effect of the tumor upon the circulation, and its pressure upon neighboring organs, the aneurism usually bursts spontaneously, a mode of termination vastly more frequent than that by recovery. If it open into the pericardium, or pleura, a genuine rupture at the thinnest point takes place; if into the œsophagus, trachea, or one of the bronchi, it gives way at some point of adhesion which forms between the tumor and one or other of these organs, and which, gradually growing thinner, at last breaks; or else a slough forms and separates, thus making an aperture into the sac. Opening into a neighboring vessel takes place after its walls have become adherent, and communication is established by a gradual wasting of the septum thus formed. More rarely, it is only the adventitia of the vessel that becomes adherent, and, after perforation at the adherent spot, blood is injected between the adventitia and media of the vessel. The external rupture of an aneurism, which has penetrated the wall of the chest, occurs either by gradual atrophy and final laceration of the integument, or else, which is most common, by loosening and detachment of a slough induced by immoderate and incessant strain.

Aneurism of the aorta is most frequently situated upon its ascending branch, before the departure of the innominata, and is more common upon its convex than upon its concave side. Those which arise outside of the pericardium, and which are generally very large, usually project toward the right half of the sternum, and become visible in the region of the upper ribs, and costal cartilages of the right side. In the majority of cases, they break into the right pleural sac, or else burst externally. Aneurisms which spring from the concavity of the ascending aorta grow in the direction of the trunk of the pulmonary artery, or toward the right auricle, which they may perforate. Those which arise from the convexity of the arch also extend to the right, forward, and upward, and make their appearance in the neighborhood of the right sterno-clavicular articulation. Those which proceed from the concavity of the arch press upon the trachea, the bronchi, and the œsophagus, which they may perforate. Aneurisms of the descending limb of the aorta often compress the left bronchus, more rarely the œsophagus, and usually open into the left pleural cavity; they may destroy the back-bone, and may come to the surface at the left side of the back. Aneurisms of the abdominal artery often attain an immense size, may also erode the spinal column, and burst into the peritonæum or retroperitoneal connective tissue.

SYMPTOMS AND COURSE.—Persons suffering from aneurism of the aorta often perish suddenly and unexpectedly of internal hæmorrhage, before the disease has given rise to any great degree of distress. At other times the symptoms are so obscure as to render a positive diagnosis impossible. In other instances, again, it admits of a more or less certain recognition from the subjective and objective manifestations which it occasions.

The signs to which aortic aneurism gives rise depend, in part, upon the crowding and compression of the adjacent organs in the thorax, as described in a previous section; and in part, also, upon obstruction of the circulation, which is one of the necessary consequences of any large aneurism.

As a result of compression of the lung or greater bronchi, dyspnœa arises, which is often extremely severe. The most intense dyspnœa, accompanied by a peculiar whistling upon breathing and coughing, attends aneurism of the arch pressing upon the trachea. If the pneumogastric nerve or its recurrent branch be stretched or irritated, the dyspnœa may assume a spasmodic, asthmatic character, and appears to proceed from the larynx, and comes on in paroxysms. Dyspnœa is one of the most common and distressing symptoms of aneurism.

A second set of signs proceeds from compression of the right auricle, the vena cava, or the vena anonyma. If the auricle or vena cava be compressed, the jugular veins swell, blue networks of veins appear upon the skin of the chest, the veins of the arms become distended with blood; indeed, it is not uncommon for dropsy soon to develop in the upper half of the body. The patient may complain of headache, dizziness, buzzing in the ears, and fits of unconsciousness may be observed. If but one of the vena innominata be compressed, the venous dilatation is limited to the corresponding side of the head and chest.

In consequence of the pressure and strain to which the intercostal nerves and brachial plexus are subjected, most violent pain often arises in the right side of the chest, the armpit, and right arm. Like most other of the troubles to which aneurism gives rise, this pain is often paroxsymal, and may deprive the patient of his sleep, and is reckoned by *Lauth* among the agents which tend to produce speedy death by exhaustion.

Compression of the arteria innominata, or of the left subclavian, may render the radial pulse extremely small or quite imperceptible. Inequality between the pulsations at the wrist of either side may also be a result of distortion of the arterial mouths, or of their stoppage by clots.

To these symptoms of compression of adjacent organs, are added those of retarded circulation. Foremost among the latter is the *pause, often so distinctly perceptible, which occurs between the beat of the heart and the wave of the arterial pulse, at a point below the aneurism.* This phenomenon is most striking when the aneurism is situated between the points of origin of the great vessels of the arch. The pulse is then felt later at one wrist than at the other; or, if the tumor involve the descending aorta, the pulse below the tumor cannot be felt until after that of the upper extremity.

As hypertrophy can only temporarily compensate the impediment to the circulation, not only does palpitation eventually set in, such as we encounter wherever the heart's action is overtasked, but the derangement in the blood's distribution, so often described, finally is established; the arteries are ill supplied, the veins and capillaries are gorged, and general marasmus and dropsy ensue. If the patient do not die of disordered circulation or of embarrassed breathing, and should he not succumb to some other intercurrent disease, the aneurism finally bursts. It would be erroneous, however, to regard this mode of termination as constant or even as the most common mode of death.

When, after coming to the surface in the form of a tumor, it breaks externally, the integument gradually grows thinner, turns dark blue, then black, and at last sloughs. After a time, the eschar separates. The blood, however, does not always escape in a stream. Sometimes it is restrained by the coagula, so that there is only a gradual trickling flow; and it may even be possible to stanch the first hæmorrhages by means of the tampon, so that death may not occur until after repeated outbursts of blood.

It is otherwise when rupture takes place into the pleura, pericardium, trachea, or œsophagus. The patient then sinks, often with extreme rapidity and in the midst of great suffering, with the signs of an internal hæmorrhage or of profuse hæmoptysis or hæmatemesis.

Life has been known to continue for some time after perforation of the pulmonary artery, or vena cava. The symptoms observed were those of extreme obstruction of the veins of the aortic circulatory system.

Although the most important symptoms of aortic aneurism are derived from physical exploration, yet dyspnœa, cyanosis, varicosity of the veins, and dropsy of the upper half of the body, severe pain in the right side and arm, inequality of pulse at the wrists, the long pause between the beat of the heart and that of the pulse at the wrist, permit us to infer the existence of this disease with great certainty. The signs vary, of course, according to the position of the tumor upon the aorta.

In aneurism of the ascending a orta, the vena cava and lungs are more especially encroached upon, so that cyanosis and dropsy of the upper half of the body, with intense dyspnœa, form the most constant symptoms.

In aneurism of the arch, it is mainly the trachea and the par vagum nerve which are compressed, and corresponding functional disturbance ensues. In many cases, also, there is dysphagia, from pressure upon the œsophagus. Inequality in the pulses at the wrists, also, is most frequent in these cases.

In aneurism of the descending thoracic aorta there is often severe pain in the back, sometimes inability to extend the spinal column, and, if destruction of the vertebræ be extensive, there may be paraplegia. There are also sometimes difficulty in deglutition and severe dyspnœa from compression of the lungs.

The functional derangements and subjective symptoms, to which aneurism of the abdominal aorta gives rise, are manifold. It may cause the most violent neuralgic pain, and, at a later period, palsy of the lower extremities, by pressure upon the nerves and erosion of the vertebræ. Compression of the digestive apparatus occasions colic, constipation, and vomiting. Pressure upon the liver and its excretory ducts may produce obstinate jaundice; while suppression of urine may result from similar action upon the kidneys. If the aneurism be situated immediately beneath the diaphragm, the latter will be pushed upward in a painful manner; and the heart will be dislocated upward and outward.

*Physical Signs.*—As long as the aneurism remains enclosed within the thorax, without touching its wall, diagnosis is not assisted by physical examination. The respiratory murmur may, perhaps, be diminished upon one side or the other, or a constant whistling sound over a compressed bronchus may be audible; but such signs admit of too many and too different interpretations to warrant our founding a decided opinion upon them.

When the aneurism touches the thoracic wall, upon *inspection*, we can almost always perceive a distinct pulsation at the point of contact, and this becomes still more evident upon *palpation*. The pulse is isochronic with the beat of the heart, or follows close upon it. It is usually stronger, also, and is almost always accompanied by a peculiar whirring "*frémissement cataire*." The point at which pulsation appears in aneurism of the ascending aorta is usually on the right border of the sternum at the second intercostal space. In aneurism of the arch it is at the manubrium sterni; in the descending aorta, it is seen upon the left side of the lower thoracic vertebræ. Where the aneurism has perforated the thoracic wall, inspection and palpation discover new symptoms. At first, one intercostal space projects in the form of a hemisphere. The tumor soon extends, admitting of no arrest of its progress. It sits firmly and immovably upon the chest, and fully conveys the impression that it has sprung from within the thorax. Sometimes the hemispherical form afterward gives place to an irregular shape. In cases of great rarity, where there is an inordinate accumulation of clot in the sac, there is no pulsation.

*Percussion* is absolutely dull and flat all over the tumor, or over the region where it lies in contact with the chest. The sense of resistance upon percussion also seems considerably increased.

Upon auscultation of aneurisms which lie in contact with the side of the chest, we hear either a murmur or a simple or a double "tone." Explanation of these symptoms is obscure.

The main cause of the systolic murmur and systolic sound is vibration of the aneurismal wall. When the vibrations, into which the latter is thrown by the entrance of the blood, are regular, a systolic sound is the result; when it is otherwise, there is a murmur. Perhaps, too, a systolic murmur sometimes arises when the aorta or pulmonary artery is compressed by the aneurismal sac, and when the blood enters the aneurism from the aorta through a narrow aperture. Diastolic sounds and diastolic murmurs are respectively the result of healthy, regular vibrations, and inefficient irregular vibrations of the aortic valves, which are transmitted to the aneurism. Generally speaking, however, even when the aortic valves are healthy, a diastolic murmur is heard above the aneurism, and not a normal diastolic sound. This, probably, is due to the recoil of a blood-wave, or to the actual regurgitation of blood from the aneurismal sac into the aorta, owing to the narrowness and roughness of the aperture of communication.

Diagnosis.—The diseases for which an aneurism is most liable to be mistaken are carcinomatous tumors of the pleura and mediastinum. Like aneurism, the latter may encroach upon the interior of the thorax, may compress and distort the adjacent organs, and, if in contact with the aorta upon one side and with the thoracic wall upon the other, may even present a circumscribed pulsating point and afterward a pulsating tumor. Distinction between the two affections is based upon the following points:

1. Carcinoma of the pleura scarcely ever appears primarily, but its occurrence is almost always consecutive to the development of cancer elsewhere, particularly after extirpation of cancer of the breast. If the etiological conditions for cancer of the pleura be absent, we may infer the existence of aneurism with great certainty.

2. Pulsation in a cancerous tumor of the thoracic wall never exhibits any lateral dilatation, while an aneurism swells up visibly with every beat.

3. A systolic murmur may proceed from pressure of a cancer upon the aorta, just as it may occur in any artery pressed upon by the stethoscope; but we never hear the double sound or double murmur in cancer of the pleura, which is so common in aneurism.

4. We rarely or never discover difference between the pulses at the wrist, when a tumor presses upon the aorta.

5. The symptoms of aortic aneurism just described are distinguished by alternate paroxysms and intervals. The symptoms of cancerous tumors, on the contrary, are steady.

The diagnosis between an aortic aneurism and an aneurism of the innominata cannot be made with certainty. The symptoms ascribed to the latter—pressure upon the vena cava superior, upon the right bronchus, the right bronchial plexus, feebleness and retardation of the right radial pulse, dulness, pulsation, and a tumor in the right sterno-clavicular region—all occur in aneurism of the aortic arch.

Prognosis.—Aortic aneurisms rarely recover. Cure has never been observed in a case where the disease has been recognized. On the other hand, life has sometimes been preserved for years, where early exhaustion of the patient has not been brought about by debilitating treatment.

TREATMENT.—Venesection repeated at short intervals, a treatment formerly much in vogue, is entirely without benefit in the treatment of aneurism. This is true also with regard to digitalis, which, like venesection, was supposed to reduce the pressure from within, and to restrain further expansion of the sac. Nor is it otherwise with the plan of placing a patient, with aneurism, upon a "vita minima;" that is, almost starving him to death, so as to reduce the volume of the blood. The action of this method, formerly much in use, can only be to aid in rendering the sufferer dropsical, and in hastening his death. The suggestion of acetate of lead, and of drugs containing tannin as a means to promote coagulation of the blood, and to fill the sac with clots, is founded upon theory alone, and deserves no reliance.

Having recognized the existence of an aneurism, we must see that the patient shun all agents which tend to increase the action of the heart. Let him live moderately; guard him from the temporary plethora which follows every excess; prescribing, however, a nourishing nitrogenous diet to counteract the threatening impoverishment of the blood.

When a tumor develops upon the wall of the thorax, and when the skin upon it begins to redden, let the patient wear a tin vessel upon the prominence, shaped according to the shape of the tumor, and filled with cold water. Electro-puncture, which has been employed a few times even against aortic aneurism, in order to produce coagulation of its contents, has hitherto had too little success to warrant repetition of the operation. To allay the pain, we are reduced to the exhibition of narcotics.

## CHAPTER III.

### RUPTURE OF THE AORTA.

THE aorta seldom bursts, if its tunics be sound. In most cases of its spontaneous rupture, its coats are the seat of the degeneration described in the previous chapter, or of the simple fatty metamorphosis mentioned while treating of aneurism. This is the case, even when an excessively-distended aorta gives way above a stricture, for then, too, the coats are almost always diseased.

In some cases the rupture at first involves the inner and middle tunics alone, while the adventitia, which is more yielding, and more easily distensible, remains for a time unbroken. The blood then flows in between the tunica media and adventitia, and forces them asunder; and thus a fusiform tumor, filled with blood, is formed, which communicates with the artery through the opening in the media and intima—*aneurisma dissecans*. According to the observation of *Rokitansky*, recovery from this condition is possible. Far more frequently, in the course even of a few hours or days, death ensues from bursting of the adventitia, and escape of the blood into the pericardium, mediastinum, or pleura. At the moment of rupture the patient sometimes suffers violent pain; but soon becomes pale, cold, and pulseless, singultus appearing with profound syncope, and other symptoms of internal hæmorrhage.

## CHAPTER IV.

### STRICTURE AND OBLITERATION OF THE AORTA.

According to *Rokitansky*, congenital narrowness of the aortic system is sometimes found, and principally in the female sex. It is accompanied by pallor, tendency to syncope, retarded development of the entire frame, but especially of the sexual organs, symptoms similar to those which attend congenital smallness of the heart.

We sometimes see a partial contraction of the aorta as a persistence of the so-called isthmus aortæ at a point between the left subclavian and the ductus Botalli. In other instances there is an obliteration of the vessel at this point instead of a contraction. In these cases we have no sufficient knowledge of the conditions occasioning the permanent narrowness, or even obliteration of the isthmus aortæ, which exists during fœtal life as a narrow communication between the arch and the descending aorta, but which becomes dilated soon after birth. It has been supposed that the ductus arteriosus Botalli might become obstructed by a thrombus, and that this thrombus might extend into the aorta; or that the ductus Botalli, while in process of obliteration, might, during its contraction, also constrict the aorta. Neither explanation is satisfactory, as both constriction and obliteration of the aorta have been observed where the duct of Botalli remained open.

The immediate consequence of contraction of the aorta is hypertrophy of the left ventricle and dilatation of that part of the aorta which lies between the heart and the point of constriction. The enormous dilatation which takes place in the branches of the subclavian, and its anastomoses with the intercostal arteries, are of great importance. The finest ramifications are converted into large branches with firm walls, and a collateral circulation so complete is set up, that the blood, in ample quantity, is conveyed around the seat of stricture into the descending aorta. The principal of these collateral channels is formed by anastomosis between the first intercostal, arising from the subclavian, and the second, which springs from the descending aorta; but extensive anastomotic communication also forms from the dorsalis scapuli, subscapularis, transversalis colli, and the intercostals. The internal mammary becomes immensely enlarged, as do the anterior intercostals which proceed from it. Its terminal branch, the superior epigastric, also dilates, and, by its communication with the inferior epigastrics, conveys blood to the iliacs.

Obliteration of the aorta, serious as the malformation appears to be, is, nevertheless, a tolerably endurable one. It may long remain latent, and the patient may attain a very great age (ninety-two years). In other instances, in course of time, palpitation of the heart, distressing pulsation of the carotids, or symptoms of hyperæmia of the brain, may manifest themselves. By-and-by a cachectic condition develops, and, in nearly half the cases reported, death has ensued with symptoms of marasmus and dropsy. Thus we see that an obstruction to circulation as grave even as that presented by obliteration of the aorta may be compensated for, temporarily, by hypertrophy of the heart, but that it finally becomes imperfect, and that symptoms then set in of retarded circulation, venous engorgement, and impoverishment of the blood, such as we have so often described. In other instances, death, by rupture of the heart or aorta, is the result, the walls, probably, always undergoing previous degeneration.

Diagnosis of this affection is based mainly upon the signs of the anastomotic circulation above described, and upon the absence of pulsation in the ramifications of the abdominal aorta. In such subjects we see varicose, worm-like, sinuous arteries, and groups of arteries, distinctly pulsating on the back along the shoulder-blade, and upon the arch of the ribs. At a point corresponding to the course of the internal mammary, we hear, near the sternum, a blowing sound, which is audible, also, at all points where the existence of dilated arteries is perceptible to sight and touch. On the other hand, in the tibials, or even in the popliteals and femorals, the pulse is feeble or even imperceptible. *Bamberger* considers that the deformity can always be recognized with certainty from these diagnostic points. Treatment of stenosis of the aorta is entirely analogous to that of stenosis of the aortic orifices, to which we therefore refer.

## CHAPTER V.

### DISEASES OF THE PULMONARY ARTERY.

ACUTE inflammation, terminating in suppuration, is quite as rare in the pulmonary artery as in the aorta. The alterations of the tunica intima, described by us as chronic endarteriitis, are often absent in this artery, where the entire aortic system is far advanced in the disease.

On the other hand, it is tolerably common even when the aorta is sound in cases of deficience of the mitral valve, with consecutive hypertrophy of the right ventricle. To it *Dittrich* attributes the frequence of hæmorrhagic infarction in the lungs of persons with heart-disease.

Aneurisms of the pulmonary artery are exceedingly rare, and never attain any considerable size. In a case observed by *Skoda* there was found in this artery an aneurism as large as a goose-egg. During life the patient had labored under signs of grave circulatory disturbance, was cyanotic and dropsical; but no diagnosis could be formed by physical exploration.

Diffuse dilatation of the pulmonary artery occurs with extraordinary frequence in cases which cause hypertrophy and dilatation of the right heart. It never produces any change in the percussion-sound over the chest (*Skoda*), but not unfrequently, and usually upon diastole, we perceive a shock, and, indeed, a distinct pulsation, in the vicinity of the root of the pulmonary artery.

While treating of metastases of the lung, we have already learned that the minuter branches of the pulmonary artery may become obstructed by the intrusion into them of wandering emboli. When one of the larger branches is thus occluded, intense dyspnœa arises, and even death may suddenly ensue from the extent of breathing-surface thus thrown out of action from interruption of its circulation. Within the last few years I have seen two cases in which death occurred in the course of a few hours, with all the signs of extreme dyspnœa and collapse, and in which it was found *post mortem* that a large thrombus had been detached from the femoral vein, had passed into the circulation, and, by obstruction of the main branch of the pulmonary artery, had occasioned this peculiar kind of suffocation. One of these cases is reported in the *Würtemberger Correspondenzblatt* by my then assistant, *Dr. Späth.*

## CHAPTER VI.

### DISEASES OF THE GREAT VENOUS TRUNKS.

It is of disorders affecting the vena cava and the pulmonary veins alone that we now treat, as diseases of the peripheral veins are treated of in the hand-books of surgery; those of the portal vein and veins of other organs are more appropriately discussed as diseases of the liver, etc.

Primary inflammation does not occur in the vena cava, and it is rare to observe inflammation and perforation of the coats of the ascending portion of it by an abscess of the liver, or of the cellular tissue behind the peritonæum. Inflammation of the pulmonary veins, terminating in abscess, is equally rare.

Dilatation of the great venous trunks takes place in diseases of the heart, which occasion engorgement of the venous system. Their constriction is almost solely the result of compression by adjacent tumors.

Primary thrombosis—that is, coagulation of the contents of a vein, with consequent inflammation of its walls—has been seen occasionally in the vena cava ascendens, but then the coagulum almost always forms first in one of the femoral veins, and afterward spreads to the vena cava. Such a formation of thrombus may be recognized by the following signs: If, in addition to the tense painful œdema of a phlegmasia alba dolens of the leg, there suddenly set in a painful swelling of the other limb, if the secretion of urine be suddenly repressed, or should it become scanty and bloody, we may infer that the thrombus has involved the vena cava and emulgent veins.

# DISEASES OF THE ORGANS OF DIGESTION.

## SECTION I.

## *DISEASES OF THE MOUTH.*

## CHAPTER I.

### CATARRH OF THE MOUTH.

ETIOLOGY.—The mucous membrane of the mouth is peculiarly exposed to the sources of injury which excite catarrh elsewhere. Hence catarrh of the mouth is a very frequent affection, but it is only recently that the name "catarrh of the mouth" has been given to those changes which, occurring in other mucous membranes, are termed catarrh. It is remarkable that this affection is rarely induced by exposure of the skin to cold, a cause which so frequently excites catarrh of other mucous membranes.

Among the injurious influences that may excite catarrh of the mouth are—

1. Irritation which acts on the mucous membrane. Dentition frequently causes catarrhal stomatitis, which often attains great severity. Rough teeth, ulcerated teeth, wounds in the mouth, very hot, very cold, or chemically injurious ingesta, smoking and chewing tobacco, etc., excite catarrh. The same effect is produced by the use of mercurial preparations, not only when mercurial salve is rubbed on the gums, or when mercurial preparations, in powder or solution, are taken by the mouth, but by inunction of mercurial ointment, and by taking mercurial pills, well covered up. For since the mercury, absorbed from the skin or the intestinal canal, is excreted by the salivary glands, it still causes direct irritation of the oral mucous membrane. Often, very small amounts of mercury will induce mercurial stomatitis; we can readily understand this, if we bear in mind that the mercurial swallowed with the saliva is again absorbed from the intestine, and eaches the mouth repeatedly before escaping from the economy. The

sensitiveness of the mouth to mercury varies with the individual; hence, in one patient stomatitis may occur sooner than in another, just as, after frictions on the skin with mercurial ointment, one person is affected earlier than another with the superficial dermatitis, which we shall hereafter describe as eczema mercuriale.

2. In many cases, oral catarrh is a propagation of inflammation from neighboring organs to the mucous membrane of the mouth. Wounds and inflammations of the face, particularly facial erysipelas, also inflammations of the fauces, are almost always complicated with oral catarrh. Less constantly, nasal and bronchial catarrh extends to the mouth. While a thickly-coated tongue was considered a certain sign of disturbance of digestion, this secondary catarrh caused facial erysipelas and angina to be almost always regarded as manifestations of gastric disturbance, and to be treated accordingly. Acute and chronic catarrh of the stomach is surprisingly often complicated with catarrh of the mouth. *Beaumont*, who had the opportunity of comparing the gastric mucous membrane with that of the mouth, in the case of the Canadian, *St. Martin*, found that changes in the former instantly excited analogous changes in the latter, and daily experience supports this observation. But, although catarrh of the mouth very frequently accompanies catarrh of the stomach, we must not suppose, on the other hand, that gastric catarrh occurs with every oral catarrh.

3. Catarrh of the mouth is not unfrequently a symptom of constitutional affection. Among the acute infectious diseases, typhus and scarlatina especially are accompanied by peculiar changes of the mucous membrane of the mouth, which are essentially catarrhal; these will be more accurately described when speaking of the diseases in question. Coated tongue is found in almost all feverish affections; but it would be going too far, to say catarrh of the mouth occurs in every fever (see treatment).

Finally, in many cases we do not know the exciting causes. *Pfeuffer* gives sitting up at night, and other observers give mental excitement, as a cause.

It is remarkable that in some patients oral catarrh obstinately persists for years, without our being able to find any continuous cause.

Anatomical Appearances.—We seldom have the opportunity of observing oral catarrh in its incipient stage. Only after severe irritation, and occasionally during difficult dentition, we see the oral mucous membrane at first dark red and very dry, till, finally, in the stage of decline, there is a copious secretion, which is clouded by containing young cells. After less severe irritation, and in the oral catarrh which usually complicates catarrh of the stomach, the intense redness and dryness of the mouth are either unobserved, or are seen only temporarily.

Quite early in the affection there is decided swelling of the mucous membrane and submucous tissue, increased secretion, and excessive formation of young cells. The swelling is most evident at the edges of the tongue and over the cheeks. The tongue appears too broad to lie between the teeth, and its sides show impressions of the teeth. A turbid mucus covers the cheeks, gums, and especially the tongue. The mucus and young cells most readily adhere to the filiform papillæ, thus giving a *coated tongue.*

*Chronic* oral catarrh has similar symptoms. The swelling of the mucous membrane is usually even more decided; on the inner surface of the lips and cheeks, and on the roof of the mouth, we not unfrequently find small nodules as large as a barley-corn (swelled mucous glands); thick yellow mucus covers the gums, especially about the teeth; the elongated processes of the filiform papillæ appear as small white threads, and give the tongue a felty or hairy look (*lingua hirsuta*). On microscopical examination (*Miquel*), it is found that the coating of the tongue, even in chronic oral catarrh, consists mostly of epithelial cells. These contain fat globules and brown granules, and not unfrequently unite together into brown plaques. At the same time we see rod-like formations, the broken epithelial processes of the filiform papillæ (*Kölliker*). Felt-like formations grow on these, their matrix forming a granular border to the hardened epithelial cells. We also find fat-globules, vibriones, and usually some remains of food.

SYMPTOMS AND COURSE.—Besides what we have said in the preceding paragraph, there is little to add to the objective symptoms. In the severe forms of acute oral catarrh, which we first described, there is a feeling of burning and tension in the mouth. Babies no longer bite the ivory ring or orris-root, which is usually given them to facilitate the cutting of the teeth. They cry when we touch their mouths, and, on attempting to nurse, they soon let go of the nipple, as if it hurt them. In some cases, whose frequency is magnified by the laity, so-called "teething convulsions" occur, which may prove fatal without leaving any material changes in the central organs to be seen on *post-mortem* examination. From our present knowledge of the subject, these convulsions must be considered as reflex symptoms, which are caused by the severe irritation of the sensitive nerves of the mouth being transferred through the central organs to the motor nerves. Indeed, it is doubtful whether these attacks are caused by the acute oral catarrh, or are the result of direct irritation of the sensitive nerves from the pressure of the teeth. (See chapter on Eclampsia.)

In moderate cases of acute oral catarrh, characterized by increased mucous secretion and excessive cellular formation, the patients complain particularly of a "bad taste," which they generally describe as slimy or clammy. Accurately speaking, this "slimy, clammy taste" is a misnomer, as physiologists only recognize bitter, sour, sweet, and salty tastes. The patients *feel* a slimy substance on the oral mucous membrane, and attempt to remove it by hawking and spitting. But the sense of taste itself is also influenced by oral catarrh. Usually the disturbance only causes the taste to be less acute, and not so sensitive. Since an insensitive layer lies between the substance to be tasted and the peripheral ends of the nerves of taste, only very irritant substances excite distinct sensations. In such cases, the patients usually say their taste is bad or stale. When they chew hard substances, and thus remove the insensitive layer from the mucous membrane, the sense of taste is for a time better. In some cases, patients suffering from oral catarrh complain of a *bitter* taste. The laity consider this a sure sign of "biliousness," and some physicians think there is a status biliosus, and not a status pituitosus. In far the greater number of cases the bitter taste is a subjective symptom; it is not excited by bitter substances, but must depend on a perversion of the nerves of taste.

Lastly, the patients not unfrequently complain of a "foul" taste; this term is also unphysiological and incomplete. This *foul* taste is caused by excitement, not of the nerves of taste, but of the olfactory nerves, with whose peripheral expansions in the Schneiderian membrane the gaseous emanations from the coating of the tongue come in contact through the posterior nares. The foul taste, or, more correctly, the foul smell, is not solely a subjective symptom; generally, other persons perceive a fetor from the mouth of the patient, especially in the morning, before breakfast; this disappears when eating has removed the foul epithelial coating from the tongue. It is doubtful whether pain in the forehead, so frequent a symptom in acute catarrh of the stomach, occurs in simple oral catarrh. The above symptoms are by no means always accompanied by disturbance of the stomach digestion. The patients often have a normal feeling of hunger; but, it is true, they usually choose very sour and salty, or highly-flavored articles of food, which can excite the nerves of taste even through the epithelial covering. Frequently there is no evidence that the stomach has not properly digested the food taken into it. After meals, there is no pressure in the epigastrium, no eructation, or other symptom of disordered digestion. It is often difficult, indeed, to persuade the patient that his stomach is sound, and not filled with decomposing substances. The thickly-coated tongue, the slimy, bitter, or foul taste, and the smell from the mouth, appear to him so distinctly to indicate an emetic, that he considers advice unnecessary.

The milder grades of oral catarrh, such as occur in most smokers, cause but slight subjective symptoms. In the morning the epithelium, which has collected during the night, usually causes a slimy taste and disagreeable odor from the mouth; but these soon pass off, and, during the day, the patients have nothing to complain of; nevertheless, they usually prefer the most piquant, to the bland and unirritating kinds of food.

In severe cases, chronic oral catarrh is a most annoying affection. The victims of it occupy a considerable time in the morning in hawking and spitting, in scraping the tongue and rubbing the teeth and gums with a hard brush to clear off the adherent mucus. The sensation, taste, and smell of the mouth are perverted all day; the odor from the mouth does not pass away. The patients consult the physician on account of the "slimy taste," for which they have in vain taken various kinds of spring water, *Strahl's* and *Morrison's* pills, and which occasionally gives them severe hypochondriasis. The healthy appearance and well-nourished state of the patients usually contrast with their complaints. On questioning them, we find that even articles of difficult digestion are eaten with impunity. It is necessary to understand these states, in order to recognize them in special cases and to treat them successfully.

DIAGNOSIS.—The coating on the tongue, observed in oral catarrh, must not be confounded with that which is found in healthy persons, especially in the morning, on the back part of the tongue, and which is called the normal coating. According to *Miquel*, this is caused by the air passing through the nose and fauces during the night, inducing evaporation from the neighboring parts of the mouth, so that the epithelium, which, under normal circumstances, is thrown off, becomes dry and forms an opaque coating. According to *Neidhart*, who, in a dissertation, written under *Seitz's* supervision, makes serious objections to this explanation of the coating of the tongue, other causes have much to do with its production. The epithelium of the mouth and tongue undergoes continuous desquamation, which is to be regarded as a result of the mechanical action of speaking and chewing. It is evident that where the movement of the tongue is greatest, and where it is most brought in contact with other parts, the epithelium will be soonest and most effectually cleaned off. Now, this occurs particularly in the anterior part of the tongue, which, with every movement, is brought in contact with the roof of the mouth, and in the sides, which lie directly against the teeth. The back part of the tongue, on the contrary, where the normal coating mostly occurs, does not lie against the roof of the mouth, and only touches it in the act of swallowing. Hence it follows, on the one hand, that the most superficial epithelial patches on the anterior part are soonest and most completely loosened, and also that they are soon removed; but, on the other hand, that they should remain attached longer at the base, and even when loosened should remain longer in position, especially as the elongated processes of the papillæ afford them a great protection.

In most feverish complaints the whole top of the tongue appears whitish. This does not generally depend on an increased formation of cells due to oral catarrh, but occurs because, while there is an increased loss of fluid through the skin, the secretions of the mouth are diminished, so that the epithelium is less moist and transparent. Besides this, patients with fever suffer from loss of appetite, and do not chew hard substances, such as best remove the epithelium. Just as, in marasmic persons with dry skin, there is continued apparent desquamation of the epidermis without its formation or removal being actually increased, so in fever the horny processes of the papillæ and the epithelium of the mouth become more evident without their being formed or removed in greater amounts. The presence of the swelling and moisture of the mucous membrance, which exist in oral catarrh, prevents our mistaking the coating of the tongue, that occurs in it, for that which we find in fevers, where the tongue is flat, small, and sometimes even very pointed, the mouth dry, and consequently the patient thirsty. If the tongue of a fever-patient is dry, without the epithelium and processes of the filiform papillæ being at least of normal amount, of course the tongue does not appear coated.

For the difference between simple oral catarrh and that accompanying gastric catarrh, see Section III., Chapter I.

PROGNOSIS.—If we except the spasms during dentition, which are sometimes dangerous to life, and whose dependence on oral catarrh is still doubtful, the prognosis as regards life is favorable. The prognosis for a perfect cure, especially in chronic oral catarrh, is less favorable, although even here a suitable, judicious treatment, if carefully followed by the patient, which is rarely done, may give a favorable result.

TREATMENT.—The causal indications cannot be fulfilled in all cases. In difficult dentition, cutting the gums is of doubtful benefit; occasionally the incisions inflame and cause the catarrh to become worse. Sharp edges of the teeth, which are easily overlooked, are to be carefully removed, wounds of the mouth and ulcerated gums are to be properly treated. Where smoking, especially the use of strong cigars, causes troublesome oral catarrh, it must be totally forbidden, or at least weak cigars only should be smoked through a cigar-holder, or, still better, a long pipe should be used. Oral catarrh, caused by the use of mercurials, requires their discontinuance, and in such cases all traces of blue ointment are to be carefully removed from the skin. The secondary oral catarrh usually disappears with the cure of the erysipelas, angina, gastritis, etc. We shall hereafter see that the latter does not require emetics nearly so often as these are ordinarily used in practice. The fact of the tongue being for a time cleaner after the vomiting depends altogether on mechanical causes, and does not at all prove that the oral and gastric catarrh are benefited. When caused by some infectious disease, the indications are the same as those for the treatment of the original affection.

The treatment of the disease itself is essentially local, just as for affections of other mucous membranes that are within easy reach. This direct treatment is especially required in those cases of chronic oral catarrh which prove very obstinate even after the exciting cause has disappeared. For this obstinate clamminess I can strongly recommend a well-known domestic remedy; namely, slowly chewing small pieces of rhubarb before going to bed. I cannot ascribe the very general improvement to the direct action of the rhubarb on the gastric mucous membrane, since it does not produce the same effect when given in very soluble pills.

In chronic catarrh persisting without cause, rinsing the mouth with solution of carbonate of soda, or slowly drinking a bottle of soda-water, on an empty stomach, is very useful. This evidently depends on the well-known power of the carbonates of the alkalies to diminish the tenacity of the mucus and render it more fluid. If this treatment is inefficacious, we may confidently order the mouth to be pencilled with a solution of corrosive sublimate (gr. j—ij, to the pound of water), as recommended by *Pfeuffer*, or with a solution of nitrate of silver (gr. j to ℥ ss water), as advised by *Henoch*. The effect of these prescriptions in oral catarrh is not inferior to that in other catarrhs from the same remedies.

## CHAPTER II.

### CROUPOUS STOMATITIS—APHTHÆ.

Etiology.—On the mucous membrane of the mouth we often see small white spots, surrounded by a red border, which look like flat vesicles; after a short time these are thrown off, and an excoriation, which heals readily, is left. This affection of the oral mucous membrane is designated by most authors as "aphthæ," a name which is also used for other diseases of the mouth, especially cancrum oris and thrush. From numerous careful observations, *Bohn* has shown that, when these white spots are punctured even in their earliest stage, no fluid can be evacuated from them, and hence that they are not vesicles, but solid thickenings, consisting of exudation on the free surface of the mucous membrane under the epithelium. I consider this view, which in the main corresponds with that of *Rokitansky* and *Foerster*, as correct; but I think that, to be consistent, the affection, which on other mucous membranes we call croupous inflammation, should have the same name when it attacks the oral mucous membrane; hence, I do not hesitate to define aphthæ as a croupous stomatitis limited to a circumscribed portion of the oral mucous membrane.

Aphthæ are chiefly observed in children. During the first few months, however, they are rarer than during dentition. Weakly, badly-nourished children are more disposed to them than strong and well-nourished ones. Among the exciting causes, cutting the teeth is the chief. Aphthæ often accompany the affection of the skin in the acute exanthemata, especially measles. Occasionally also they occur as small epidemics, without any perceptible cause, and it then appears as if they spread by contagion. Lastly, aphthæ accompany other severe affections of the mouth, particularly cancrum oris.

ANATOMICAL APPEARANCES.—Aphthæ chiefly occur on the anterior half of the tongue, and the inner surface of the lips, cheeks, and hard palate. They are about the size of a lentil, round, often quite numerous, and are either disseminated or run together into irregular figures. The grayish or yellowish-white deposits separate gradually from the periphery toward the centre, making the red border broader. When the exudation has entirely separated, there is no ulceration, only an excoriation; this is characteristic of a croupous affection, and distinguishes it from a diphtheritic. The excoriated place is soon covered with epithelium again; aphthæ leave no cicatrix. Catarrh, with copious production of mucus and cells, affects the rest of the mouth.

SYMPTOMS AND COURSE.—The eruption of aphthæ is often preceded for several days by fever, restlessness, loss of appetite, and the symptoms of oral catarrh. The disease itself is accompanied by pain, which is increased by nursing, and in older children by speaking and chewing. At the same time the secretion of saliva is so much increased, that a clear fluid almost constantly runs from the half-open mouth. From the decomposition of the accumulated epithelium and the exudation thrown off, there is a disagreeable, penetrating fetor from the mouth, especially in the not unfrequent complication of the croupous with diphtheritic stomatitis (see Chapter III.). By repeated relapses the affection may last several weeks. Of itself it is scarcely ever dangerous.

TREATMENT.—Chlorate of potash has very properly attained the reputation of a specific for aphthæ. In almost all cases, under the use of a watery solution of this remedy (gr. jv—vj. at a dose), improvement and cure very soon occur. If, contrary to our expectation, the chlorate of potash does not produce this result, we may paint the aphthæ with dilute muriatic acid, or with nitrate of silver.

## CHAPTER III.

### DIPHTHERITIC STOMATITIS, STOMACACE, CANCRUM ORIS, MUNDFÄULE.

ETIOLOGY.—As has been repeatedly said, in diphtheritic inflammation a fibrinous exudation is deposited in the tissue of the mucous membrane, and the part of the membrane affected sloughs from the compression to which its vessels are subjected by exudation. After the detachment of the diphtheritic slough thus formed, which is sometimes dry, sometimes moist, a loss of substance remains.

Diphtheritic stomatitis results—1. From the too continued or too excessive use of mercurials. 2. It not unfrequently occurs without perceptible cause, especially among people living under unfavorable circumstances (mal logés, mal vêtus, mal nourris, *Taupin*, *Bohn*). The latter form is usually called stomacace or cancrum oris; extensive epidemics of it occur in foundling hospitals, orphan asylums, barracks, and other institutions, and also in armies not in barracks, but in the field, or otherwise living in the open air; it is not improbably extended by contagion.

ANATOMICAL APPEARANCES.—In the milder grades of the diphtheritic form of mercurial stomatitis, at certain parts of the mouth, along the lateral borders of the tongue, and on the parts of the cheeks and lips which lie against the teeth, we at first find a whitish or somewhat dirty discoloration of the mucous membrane. These white spots cannot be wiped off, but after a few days the superficial layer of mucous membrane, with the exudation infiltrating it, falls off, and in its place is left an unhealthy-looking ulcer, which cleans off slowly and finally cicatrizes from the margins. In more severe cases, where the exudation infiltrates and destroys the whole thickness of the mucous membrane, a large portion of the inner surface of the mouth is often converted into a soft, discolored slough. If this separates, a deep ulcer with irregular borders and uneven base is left. The loss of substance is but slowly filled with granulations, and as the lost mucous membrane is not regenerated, but is replaced by cicatricial tissue, contracted cicatrices, or even adhesions and false anchylosis, not unfrequently remain.

In cancrum oris the infiltration and ulceration always begin on the gums, usually at their upper border on the anterior surface. In severe cases it advances to the posterior surface of the gums, and the adjacent parts of the lips, cheeks, and tongue. The teeth become loosened, and occasionally the periosteum of the jaw is exposed and destroyed. In consequence of this, in some cases there are caries and necrosis of the maxillary bones.

SYMPTOMS AND COURSE.—The diphtheritic form of mercurial stomatitis is accompanied by severe pain, particularly when the sloughs have separated and left ulcers. Chewing, or even speaking, will render this pain unbearable. The secretion from the salivary and mucous glands is enormously increased, the patients cannot sleep, as the secretion, if not ejected, runs into the larynx, and induces cough or suffocation; if they lie on the side and succeed in sleeping, they soon wake to find the pillow cold, wet, and saturated with saliva. On the parts of the tongue and gums, but especially along the edges of the teeth, which are free from sloughs or ulcers, there is an unusually thick, yellow, soft coating. There is a very penetrating bad odor from the mouth, caused by the decomposition of this coating, and of the slough of the mucous membrane. It is not decided whether or not this smell is caused by the formation of sulphuret of ammonium from the disintegration of the sulpho-cyanide of potassium, which is a normal constituent of the saliva, by the decomposition going on in the mouth. Even when the mercurials have been stopped, the pain, flow of saliva, and smell, pass off slowly, and even in mild cases it is usually from eight to fourteen days before the patient feels well. In severe cases the cure progresses even more slowly; as was mentioned above, there may even be permanent injury.

At the commencement of cancrum oris, according to the excellent description of *Bohn*, the gums are dark red and greatly swelled by excessive hyperæmia. They appear loosened from the teeth, and bleed on the slightest pressure. After this stage has lasted two to four days, a gray membranous deposit almost always appears on the upper border of the gums, particularly about the incisor teeth of one side of the lower jaw. On careful examination, we find that this deposit, which is a pulpy substance, does not lie on the gums, but consists of the gangrenous tissue of the gums. After the separation of the pulpy mass which we, in opposition to *Bohn*, must consider a diphtheritic slough, there is found a loss of substance of the gums, on the surface and periphery of which the same process is repeated as long as the affection lasts, until in severe cases the contours of the gums are lost, the teeth loosened, and the other evils described above have resulted. At the same time the neighboring lymphatic glands are swelled and painful; the parts of the lips and cheeks corresponding to the affected part are œdematous; the breath has a cadaverous odor; the saliva, which is often bloody and discolored, flows constantly from the mouth; and every attempt to swallow, or even to drink, causes severe pain. The patients avoid closing the mouth and even keep the jaws apart, to prevent rubbing. Strange to say, the general condition and even the appetite are affected but little; fever is slight or even absent; when properly treated, the disease almost always runs a favorable course, the diphtheritic sloughs separate, and the ulcers under them heal in a relatively short time. Neglected and improperly treated, the affection may exist for months, but it rarely endangers life. Fatal results probably always depend on complications.

TREATMENT.—In the diphtheritic form of mercurial stomatitis we must not neglect to tell the patient how slow his cure will be. He will bear his sufferings much better if he does not find his hopes disappointed from day to day; but if we promise him that, if not cured sooner, he will, at least, be comfortable by the eighth or ninth day, he will be patient and submit to what is unavoidable. Frequently washing the mouth with cold water, or with water and red wine, at the commencement, subsequently painting the ulcers with dilute muriatic acid, or, still better, with the solution of nitrate of silver, described in Chapter I., but especially the use of solution of chlorate of potash, are far preferable to the internal administration of iodide of potassium and mercurial antidotes, or painting the mouth with spirits of camphor, which is as painful as useless. Touching the ulcers occasionally with solid nitrate of silver is very beneficial, but exceedingly painful.

Chlorate of potash is just as certain a specific against cancrum oris as against aphthæ. To children under one year we may give one scruple daily, to older ones half a drachm, to adults one to two drachms, dissolved in six ounces of water. Under this treatment the bad odor very quickly disappears; the ulcers also commence to clean up in a few days and heal rapidly. We are very rarely obliged to touch the ulcers with nitrate of silver.

## CHAPTER IV.

### EXCORIATIONS AND ULCERS OF THE MOUTH.

ETIOLOGY.—The rapidly-healing excoriations which remain after aphthæ, and the ulcers caused by mercurial stomatitis and cancrum oris, were discussed in the last chapter and the one before it.

Small vesicles, followed by very painful excoriations about the point of the tongue, appear to be caused by local injuries; at least, the patients suffering from them always say they must have burnt themselves, smoked too much, etc.

Diffuse catarrhal ulcers rarely occur in the oral mucous membrane; still I have observed them in some cases. In one instance the greater part of the surface of the tongue was the seat of an obstinate catarrhal ulceration.

Follicular ulcers not unfrequently occur from stoppage, swelling, and ulceration of the large mucous glands, which are particularly plentiful on the inner surface of the lips. In some females these almost always occur at the menstrual periods; in others, during pregnancy or lactation (stomatitis vesicularis materna). They are also seen in men without any apparent cause.

Irregular ulcers at the angles of the upper or lower jaw occur quite often. According to *Bednar* and *Bamberger*, they result from the destruction of a fibrinous exudation, infiltrating the mucous membrane, but by their limitation to the above-named locality are distinguished from the ulcers left by cancrum oris.

Variolous ulcers result from the eruption in variola passing from the skin to the mucous membrane of the mouth. Herpetic vesicles may attack the mouth and cause small herpetic ulcers. The callous ulcers on the tongue caused by sharp edges of teeth, and those of the gums resulting from the formation of tartar, belong to surgery. Syphilitic and scorbutic ulcers will be treated of in separate chapters.

ANATOMICAL APPEARANCES.—The small vesicles and excoriations at the end of the tongue are only discovered on careful examination. If the vesicle has ruptured, it looks as if the epithelial processes of one or more filiform papillæ were broken off; we see only a small, slightly-excavated red spot. In diffuse catarrhal ulcers, there is a loss, not only of the epithelial covering of the mucous membrane, but also a superficial loss of substance of the mucous membrane itself, of variable extent, and usually of irregular shape. The rest of the mucous membrane of the mouth shows the above-described changes of catarrhal inflammation with extensive production of cells.

Follicular ulcers are rarely numerous; frequently there is only one. This usually starts as a bright, pearly vesicle, which subsequently breaks, and becomes an oval ulcer, several lines long. The base of this ulcer is quite yellow, or lardaceous, and covered with a thin secretion; the edges are somewhat elevated, hard, and red.

The irregular ulcers at the angle of the jaw are occasionally symmetrical on both sides; they may be several lines long, are irregularly shaped, and present a loss of substance of the mucous membrane, which even extends into the submucous tissue. They often cause obstinate swellings of the cervical glands.

The variolous ulcers occur particularly on the roof of the mouth. After the rupture of the flat pustules, with which the eruption begins, superficial, round, easily-healing ulcers are left. Herpetic ulcers usually occur on the insides of the cheeks, and on the roof of the mouth. The vesicles, which form groups like the herpes vesicles on the lips, break early, and leave flat ulcers, which soon heal.

SYMPTOMS AND COURSE.—The small vesicles and excoriations on the point of the tongue are annoying, but perfectly free from danger. They disappear without treatment in a few days. The inconvenience they cause contrasts strongly with the very slight anatomical changes.

The diffuse catarrhal ulcers render the motions of the mouth, especially of the tongue, very painful. After they have lasted some time, this pain seems to diminish, even if the objective symptoms continue unchanged.

Follicular ulcers also are accompanied by pain in speaking and chewing, and by the symptoms of oral catarrh. The lardaceous base and hard edges of these ulcers greatly frighten half-initiated non-professionals, who have suffered from syphilis, because, from these appearances, they make up their minds that the ulcers are of specific nature.

The ulcers at the angle of the jaw render chewing and swallowing difficult, and, in some patients, cause severe pain, while in others they are not discovered till the mouth is carefully examined. They are rarely dangerous, although they sometimes last for weeks.

Variolous and herpetic ulcers rarely cause much pain.

TREATMENT.—The small vesicles and excoriations at the point of the tongue disappear if the mouth is kept free from injuries for a few days, smoking and the use of hot food, etc., being avoided.

Chlorate of potash is not so serviceable in diffuse catarrhal ulceration as in other affections of the mouth. Continued and energetic local treatment with nitrate of silver, and particularly with a weak solution of corrosive sublimate, is most serviceable. The beneficial effects of the latter are analogous to those attained by the treatment of some skin affections with mercurials.

In treating follicular ulcerations, we must look out for any disturbances of digestion. If these have been removed, or are not discoverable, we confine ourselves to the use of chlorate of potash and energetic local treatment. Touching the ulcer with solid nitrate of silver is very painful, it is true, but it acts surely and quickly.

The ulcers at the angle of the jaw require no internal treatment, but chlorate of potash is recommended for them also. Locally, we may use nitrate of silver or concentrated acetic acid, as recommended by *Rilliet* and *Barthez*.

Variolous and herpetic ulcers require no special treatment.

## CHAPTER V.

### SYPHILITIC AFFECTIONS OF THE MOUTH.

ETIOLOGY.—*Primary* ulcers and condylomata—that is, those developing at the point where the transfer of the syphilitic poison has taken place—occur, according to my observation during the last few years, much more frequently than I had formerly supposed. In some cases the contagion passes from the nipple of a syphilitic nurse to the mouth of the nursling. Occasionally the infection is caused by unnatural debauchery; most frequently through so-called sugar-teats, which pass from the mouth of a syphilitic to that of a non-syphilitic person. From one town in the vicinity of *Tübingen*, I have treated, and shown in the clinic, a family of ten persons, children, parents, and grandparents, who all had syphilitic ulcers and condylomata of the oral mucous membrane from this cause.

Among the *secondary* syphilitic affections (by which term we mean those that occur in the early stages of the disease only, not at the point of infection, but at other parts of the body), condylomata and ulcers often occur together in the mouth. Among the *tertiary* forms (those which occur in the later periods), we have the gummy tumors, or nodular syphilomata (*Wagner*) of the tongue, which are often mistaken for cancer of the tongue.

ANATOMICAL APPEARANCES.—Both the primary and secondary syphilitic ulcers and condylomata spring from circumscribed indurations, or from syphilitic papules of the mucous membrane. Then an excessive collection and milky cloudiness of the epithelium give a peculiar white appearance (as if it had been touched with nitrate of silver) to the surface of the affected part. Subsequently the papules of the mucous membrane form syphilitic erosions or ulcerations by molecular disintegration, or condylomata by papillary proliferation, or both together. The ulcers occur most frequently at the corners of the mouth; here they are usually superficial, and it looks as if the commisure of the lips were torn. On the edges of these ulcers there are almost always small condylomata. The ulcers occurring on the tongue, especially on its upper surface or sides, which are exposed to many sources of injury, form more or less deep fissures, or extensive losses of substance, whose uneven base is covered with a whitish-gray detritus. On the lateral edges of the tongue, the condylomata usually form elongated, shallow excrescences; on the dorsum of the tongue, on the contrary, they form round or oval warty vegetations, with broad bases, often separated by fissures. Not unfrequently, patients who have a bad conscience, and occasionally, also, inexperienced physicians, mistake the circumvallate papillæ at the base of the tongue for syphilitic condylomata.

Gummy tumors, or nodular syphilomata of the tongue, usually come on the anterior third. At first only an indurated spot is noticed; this soon swells to the size of a bean or hazel-nut, and subsequently softens and ruptures. After the rupture of the nodule there is left a deep, sharply-bounded ulcer, with inverted, thickened borders.

SYMPTOMS AND COURSE.—The primary and secondary ulcers in the mouth cause pain in chewing and speaking, and are accompanied by the symptoms of chronic oral catarrh, described in the first chapter. The diagnosis rests partly on the history, partly on the objective symptoms above given.

When condylomata occur at the edges of the tongue, they cause little annoyance, and would be easily overlooked, if patients who have long suffered from syphilis did not pay such attention to themselves. They often recede at one place, while new ones come at another. In other cases, without any treatment, they disappear for a longer or shorter time, but come back again, and, with any treatment, show great tendency to relapse. Condylomata on the dorsum of the tongue impede its movements, and thus become annoying. Inspection of the mouth renders the diagnosis certain, as the affection is not easily mistaken.

The nodular syphilomata of the tongue develop without pain, and even their ulceration does not cause much, but they render the tongue unwieldy and rigid, and thus interfere with speaking and chewing. But the ulcers left, after their breaking down, are very sensitive to the touch of the teeth, and to hard articles of food.

TREATMENT.—The principles for treating syphilitic affections of the mouth will be given hereafter.

The effect of preparations of mercury on primary and secondary ulcers and condylomata of the mouth is, as a rule, very striking. We may, almost with certainty, reckon that, under mercurial treatment, they will very shortly improve, and entirely disappear. Nevertheless, we must guard against the misuse of mercurials, especially in repeated relapses of syphilitic papules of the mouth, when they are the sole symptoms of syphilis. Even the nodular syphilomata of the tongue may disappear at any stage, under proper treatment.

## CHAPTER VI.

### SCORBUTIC AFFECTIONS OF THE MOUTH.

ETIOLOGY.—The affection of the gums is among the most constant and among the first symptoms of scorbutis. The changes in the gums are exactly analogous to those caused by the disease in other tissues.

They compel us to suppose an abnormal condition of the capillary walls, which explains the various exudations and inclination to hæmorrhage, seen in scorbutis, better than would be done by an abnormal condition of the blood, the nature of which is entirely unknown, and whose presence even has not been proved.

For the causes of scorbutis, and hence almost always of this affection of the gums, see chapter on scorbutis.

ANATOMICAL APPEARANCES.—The seat of the scorbutic affection of the mouth is exclusively in the gums, but, where any of the teeth have been lost, the gums are just as free from the affection as other parts of the mouth, and, where all the teeth are gone, the patients do not have any scorbutic affection of the mouth. Occasionally the affection is limited to one side, and in some cases to the parts around a few teeth. At the commencement there is a red border to the upper margin of the gums; these soon begin to swell, and to become dark blue. The pointed processes between the teeth especially swell out, and their attachment to the teeth is loosened. The swelling, which depends on œdema and the escape of blood into the parenchyma of the gums, may become so great that the gums press over the teeth and hide them, or that spongy swellings, half an inch or more thick, form on the gums. About the teeth, and at the top of the swelling, the surface subsequently disintegrates to a soft, discolored mass, after the separation of which there is left a loss of substance. This sloughing appears to be caused partly by the excessive tension of the infiltrated portion, partly by the pressure it is subjected to by the teeth. When improvement begins, the swelling of the gums subsides; they again become attached to the teeth, and finally attain their normal color. In a few cases, a new formation of connective tissue seems to occur during the affection; after the swelling has subsided, the gums retain a cicatricial solidity, are uneven and nodular.

SYMPTOMS AND COURSE.—Chewing is very painful, and often impossible, on account of the swelling of the gums. The secretion of mucus and saliva in the mouth is greatly increased. Hæmorrhages occur on attempting to chew, as well as from slight pressure on the gums. The decomposition of the contents of the mouth, which are mingled with blood, and subsequently with dead tissue, causes a very penetrating and disagreeable odor. These symptoms, and the examination of the mouth, in which we find the above-described changes, together with the observance of the other symptoms of scorbutis, confirm the diagnosis of a scorbutic affection of the mouth.

TREATMENT.—With proper treatment of the original affection, the affected gums often return to a normal state in a surprisingly short time. Along with the dietetic and therapeutic remedies for the original disease, which will be described hereafter, it is customary to use astringent mouth-washes, such as spiritus cochleariæ, tinctura myrrhæ, rhataniæ, or decoctions of willow, oak, or Peruvian bark. In an epidemic in Prague, observed and described by *Cejka*, washing the mouth with warm vinegar, containing more or less brandy, was found beneficial where there was loosening of the gums. Where the affection of the gums was more severe, a linctus, with muriatic acid, was prescribed. Subsequent relaxation was treated by astringent decoctions and solutions of alum.

## CHAPTER VII.

### SOOR—MUGUET—THRUSH.

ETIOLOGY.—Thrush was considered as a peculiar form of exudative stomatitis until it was discovered that a parasitic plant, growing on the mucous membrane of the mouth, caused the disease, or at least had a great deal to do with it. The thrush-fungus, oidium albicans (*Robin*), is not found outside of the organism, hence we do not know how its germs reach the mouth. But there is no doubt about certain circumstances being required for the germs to reach the mouth and the fungus to grow there. In children the affection is only seen during the first days and weeks of life, rarely in the second month; in adults it only occurs in protracted, exhausting diseases a short time before death. Hence it appears probable that the germs become attached most readily, and the fungus grows best, where the acts of chewing and swallowing are not very energetically performed, and the fungus can remain quiet and find nourishment in the disintegrating product of the epithelium and remains of food adherent to it. The layer of mucus, lining the mouth, appears to interfere with the implantation of the fungus. In the newly-born or in moribund patients we may often foretell appearance of the thrush fungus, from a certain amount of dryness of the mouth. It is not probable, at least for all cases, that the diminished secretion from the mouth is due to the first stage of catarrh, even though the unaccustomed irritation in the delicate oral mucous membrane of a newly-born child may readily cause catarrhal irritation. Neglect of cleansing the mouth greatly favors the development of thrush. In large lying-in and foundling hospitals, where the care, by which thrush can almost always be prevented, is not exercised, the affection often attacks almost all the children. Although thrush has, in some cases, been transferred directly from the oral mucous membrane of one person to that of another, still the affection cannot be reckoned among those that spread by contagion. It is not necessary for a patient with thrush to be in the vicinity, for another to be affected with it; but the germs of thrush, like those of mould, seem to be very plenty and widely-spread, and to develop wherever they find a suitable place and favorable circumstances.

ANATOMICAL APPEARANCES.—Whitish points, or a delicate frosty coating, in severe cases a cheesy, smeary mass, looking much like curdled milk, are found on the inner surface of the lips, tongue, and roof of the mouth. At first these may be readily removed, subsequently they are more firmly attached. From the mouth the coating occasionally advances to the larynx, more frequently to the œsophagus; in some cases the latter has even been found filled with thrush deposit. The disease scarcely ever extends to the stomach. On microscopical examination of the milky deposit it is found to consist of young and old epithelial cells, fat-globules, etc., between which peculiar round granules and filaments may be seen. The first are known to be spores of fungi by their oval form, sharp outlines, by the excavation which is evident in the larger ones, and by their difference in size, indicating their growth. The filaments starting from the spores are of variable thickness; they have sheaths and constrictions; where the latter occur they have branches, which go off from them at acute angles, and have the same calibre as the trunks. These fungoid filaments form beautiful tree-shaped figures, or, when very numerous, form a thick felt. It often looks as if they perforated the epithelial cells, located at their joints. At first the thrush-spores are in the most superficial layers of the epithelium; later they press in between them. They may even, although this rarely occurs, grow into the mucous membrane itself.

SYMPTOMS AND COURSE.—Children suffering from thrush almost always show that nursing pains them. Patients dying of phthisis, carcinoma, etc., complain of painful burning in the mouth, when they are affected with sprue. It is uncertain whether these difficulties depend on the injuries that the deposit causes to the mucous membrane, or whether a coincident slight inflammation of the oral mucous membrane excites these pains and causes the growth of the fungus. In children, suffering from thrush, we often see diarrhœa, accompanied by pain in the abdomen, with fluid, green, acid stools. At the same time there are not unfrequently redness and excoriations about the anus, inside of the thighs and nates. As this symptom also occurs in nursing infants without thrush, and as many children having thrush have no diarrhœa, many consider it as an accidental complication, while others, especially French observers (*Valleix*), have considered diarrhœa among the symptoms of thrush. It is difficult to decide which is right. In many cases the diarrhœa may be independent of the thrush. But, as we have to ascribe so many cases of infantile diarrhœa to abnormal decomposition of the ingesta, and as we know that, where microscopical germs form, there is usually abnormal decomposition, it does not appear very improbable that part of these diarrhœas are caused by the presence of thrush-deposit in the mouth and its passage into the stomach and intestine.

TREATMENT.—Even sensible and attentive mothers rarely use an amount of care in cleaning the baby's mouth sufficient to prevent the development of thrush. It is true the mouth is washed out in the morning while bathing the baby, and at night when undressing it; but during the day they let it go to sleep on the breast, carefully withdraw the nipple from the mouth so as not to awake it, and lay it in the cradle, while the last portions of milk, not yet swallowed, remain in the mouth and decompose, preparing the mouth for the thrush-fungus. At the same time most nurses, partly from ignorance, partly to excuse their neglect, conceal from the mother the fact that neglect of cleanliness is the cause of the development of the fungus. They even designate the sprue as a "healthy affection," which is advantageous for the well-being of the child. The physician should most strongly urge his patients to carefully wash the baby's mouth with a linen rag dipped in water, or a mixture of water and wine, after each feeding, whether it is to go to sleep or not. If this rule is carefully followed, the children will almost certainly remain free from thrush.

Even after the development of thrush we may limit ourselves to carefully removing the creamy deposit and washing the mouth. The home remedies, such as sprinkling the mouth with sugar, painting it with borax and mel rosæ, which are advised by the nurses, are to be avoided; they render the mouth sticky, give new material for decomposition, and do not at all interfere with the development of the thrush. The accompanying diarrhœa must be treated according to principles hereafter set down.

## CHAPTER VIII.

### PARENCHYMATOUS INFLAMMATION OF THE TONGUE—GLOSSITIS.

ETIOLOGY.—In most cases of glossitis an exudation is deposited between the muscular filaments of the tongue while they themselves are rarely inflamed or destroyed (see pathogenesis of myocarditis).

Acute parenchymatous glossitis is a rare affection, it is only induced by severe injuries affecting the tongue; such as burns and injuries from acrid or caustic substances, and especially from bee and wasp stings. Chronic partial glossitis results most frequently from the pressure of sharp edges of the teeth and rough pipe-stems. We do not know the causes of dissecting glossitis.

Anatomical Appearances.—In acute glossitis the whole tongue is usually affected; very rarely the affection is limited to one side. The tongue often appears doubled in size, dark red, the surface smooth or fissured, and covered with tough, often bloody exudation. The substance of the tongue is infiltrated, softened, and pale. When resolution occurs, the tongue returns rapidly to its normal size and structure; in other cases it remains for a long while, or permanently, somewhat indurated and enlarged. In severe glossitis, small abscesses, filled with purulent fluid, form in the substance of the tongue; these enlarge, unite, and may perforate the mucous membrane, and be evacuated. The loss of substance thus caused leaves a radiated, depressed cicatrix when it heals.

In chronic partial glossitis we find, particularly at the edge of the tongue, circumscribed hard spots, which project slightly or not at all, and which often retract the neighboring parts of the tongue just like cicatrices. At these points the muscular substance has disappeared, and is replaced by connective tissue.

In the form called *glossitis dissecans*, the tongue is divided into lobules by deep furrows over its surface. Remains of food and epithelium collect in these furrows and cause ulceration. Many apparent cracks in the surface of the tongue are simple folds of the mucous membrane, and are analogous to the wrinkles of the skin, which we so often see in old persons, especially in the face.

Symptoms and Course.—In acute glossitis there is not room enough in the mouth for the enlarged tongue, which projects almost an inch beyond the teeth, which are kept apart. The upper surface is whitish, or, if the exudation covering it is mixed with blood, it is dirty brown; the under surface is dark red. The deep impressions made by the teeth in the sides soon change to ulcers with fatty coatings. The tension of the tongue caused by the great swelling excites severe pain. The movements of the tongue are impaired by the pressure caused by the exudation on the muscular fibres, speech becomes unintelligible and soon impossible, chewing and swallowing the same way. The saliva constantly runs out of the mouth at both sides of the tongue, while, the mouth being open, evaporation constantly goes on, and the surface of the tongue, not being moistened, becomes dry and incrusted. The submaxillary and the lymphatic glands of the neck enlarge, the circulation in the jugular vein is obstructed; the face appears blue and swollen. The entrance to the larynx may be contracted by the swelling at the root of the tongue, and respiration be very much impaired; hence attacks of suffocation often occur at the height of the affection; these may cause death. Acute glossitis is accompanied by high fever, full pulse, great anxiety and restlessness, and severe constitutional disturbance; but, when the breathing has been affected for some time, the symptoms change: the pulse becomes small, the patient listless, and the symptoms occur that we have described as caused by carbonic-acid poisoning. When the disease runs an entirely favorable course, these symptoms gradually subside; they are often suddenly arrested by suitable treatment. When abscesses form, all the symptoms increase; but, when perforation occurs, they subside almost instantly.

Chronic partial glossitis causes a circumscribed dull pain, which only becomes burning when there is coincident ulceration of the mucous membrane. The induration impairs the movement of the tongue. The affection may last for years, and is often mistaken for cancer of the tongue.

Glossitis dissecans is very painful as long as there is any excoriation between the divisions of the tongue. When these have healed, abnormal divisions of the tongue remain, but do not incommode the patient.

TREATMENT.—The great danger of acute glossitis requires that the treatment should be very energetic. General bleeding, leeches or cups to the neck, are of no use. Leeches applied directly to the tongue increase the evil. Blisters to the nape of the neck, determination to the intestines by the use of laxatives or stimulating enemata, are just as useless. It is much better to scarify the whole top of the tongue boldly and deeply; owing to the swelling, we need not fear wounding the ranine artery. When the disease is at its height, we may give the patient pieces of ice in the mouth, and not give soothing mouth-washes till the symptoms have moderated or the glossitis subsided. If deep incisions are ineffectual and suffocation threatens, tracheotomy may be necessary.

In chronic partial glossitis we must first of all remove the sharp edges of teeth, etc. But frequently this is insufficient, and operation is the only effectual treatment. Iodine, water from mineral springs, and systematic purgation, have been recommended on theoretical grounds; experience has not proved their advantage.

In glossitis dissecans we may limit ourselves to the treatment of the ulcers by nitrate of silver in substance or solution.

## CHAPTER IX.

### NOMA—WATER CANKER—GANGRENOUS SORE MOUTH.

ETIOLOGY.—Noma is that form of gangrene which results from an asthenic inflammation, that is, from an inflammation occurring in a debilitated person. "If a nutritive change of destructive character affects parts which have been greatly altered by previous changes of their nutrition, entire death of the part may quickly result." (*Virchow.*)

The disease is almost exclusively encountered among children, especially among those who have become cachectic from want of care, insufficient or spoiled food, and bad dwellings; or among those who have just recovered from severe illness that has greatly weakened them. Noma is most frequently seen as a result of measles, more rarely after other acute exanthematous affections, or after typhus, pneumonia, etc. Misuse of mercurials in the treatment of the above affections appears to have much to do with the occurrence of noma as a sequel; it often begins simultaneously with mercurial stomatitis. In the north of Germany, and especially in Holland, it is more frequent than in the south. It seems never to be epidemic.

ANATOMICAL APPEARANCES.—The affection almost always begins on the inside of the cheeks. Over a spot hardened by infiltration, the mucous membrane becomes red, then discolored; a vesicle filled with cloudy serum, often forms on this. The affected part soon blackens, softens, and disintegrates. The gangrene spreads, destroys the gums, the lips, the base and edges of the tongue on the affected side; the maxillary bones are exposed and exfoliate, the teeth become loose and fall out. Progressing, the gangrene reaches the outer surface of the cheeks, spreads rapidly, and finally changes the entire cheek, part of the nose, the lower eyelid, often even half the face, into a ragged, pulpy, moist mass, or to a dry, black slough. The blood-vessels resist the destruction longest; on *post-mortem* examination, they are found still preserved, but filled with fibrinous coagula. In the few cases that recover, the gangrenous masses are thrown off and the loss of substance is filled with granulations, so that a firm, fibrous cicatrix finally results from a new formation of connective tissue. Adhesions in the mouth and frightful disfigurement always remain.

SYMPTOMS AND COURSE.—According to the excellent description of *Rilliet* and *Barthez*, while the gangrene commences, usually without pain, on the inner surface of the oral mucous membrane, a soft, regular, circumscribed œdema occurs in the affected cheek and lip, and gradually spreads. A hard, round nucleus forms in its centre, over which the skin appears shining, pale, or mottled violet. Even when the inside of the cheeks and a great part of the gums have become gangrenous, the child often sits quietly in bed. A sanguineous, or even black saliva, runs out of his mouth; but he plays, demands food, takes it eagerly, and with the food swallows the sloughs that fall off from the gangrenous parts. At the same time the skin is pale and cool, the pulse small and moderately frequent, and there is delirium at night. Occasionally, mostly at the fifth or sixth day of the disease, a circumscribed, dry, black slough forms on the cheeks or under-lip; this increases daily, till it affects half the face. Occasionally, even at this stage, the child is tolerably strong, demands food, and tears gangrenous pieces out of the mouth. The appearance becomes more hideous when the slough separates, and tags hang from the cheeks, between which we can see the bare, loosened teeth, and blackened jaw-bone. Then the smell is excessively disagreeable, the patient very weak, and diarrhœa usually comes on; thirst is almost unquenchable; the skin is cool and dry; the pulse small and imperceptible; finally, the child dies of exhaustion. Occasionally, the disease begins to recover from the first stage; but, even after detachment of the external slough, the gangrene may be limited, the swelling diminish, the general health improve, the surfaces of the wound clean off, and healthy suppuration occur.

TREATMENT.—Quinine, chlorine-water, charcoal, and other antiseptics, have been recommended as internal remedies; but they are of little service; they are recommended more on theoretical grounds than from experience of their benefit. We should give the patient fresh air, good nourishment, a small amount of wine, and treat the gangrene locally, according to surgical principles. Almost all caustics have been advised for noma; the actual cautery has obtained the greatest reputation. The object of these applications is to destroy the gangrenous parts, and to excite inflammatory reaction in the surrounding parts.

## CHAPTER X.

### PAROTITIS—INFLAMMATION OF THE PAROTID AND ITS VICINITY—MUMPS.

ETIOLOGY.—Besides the cases caused by wounds of the parotid, by the entrance of foreign bodies into its excretory duct, or by calcareous deposits, which cases belong to the surgeon, we distinguish two forms of parotitis: 1. Idiopathic parotitis, parotitis polymorpha (mumps); 2. Symptomatic or metastatic parotitis.

In opposition to the generally-received opinion, *Virchow* maintains that the affection starts in the gland-ducts of the parotid. He has directly proved this in the case of symptomatic parotitis, and in the idiopathic form also it appears to us much more probable that the inflammation should begin in the gland-ducts than in the interstitial tissue. If, with *Virchow*, we consider idiopathic parotitis as resulting from a simple catarrh, which has no tendency to suppurate, and the symptomatic or metastatic form, as caused by catarrhal inflammation of the gland-ducts, that *has* a tendency to suppurate, the symptoms, course, and so-called metastases of the affection are less inexplicable than if we follow the old view, which considered the intercellular substance of the gland as the starting-point and peculiar seat of the affection.

*Idiopathic parotitis* is rarely sporadic; it almost always occurs in epidemics; these usually come in the spring and autumn, that is, in cold, damp weather, rarely in the dry, warm weather of summer. They vary in duration and extent; occasionally they are confined to certain institutions, foundling houses, barracks, etc. Trustworthy observations render it most probable that the disease spreads by contagion. It does not appear to us justifiable (with *Rilliet*) to consider mumps as an infectious disease, and the inflammation of the parotid as the local expression of a constitutional disease, and to regard it as analogous to the affections of the skin that accompany the acute infectious diseases. The same objections that we have raised to considering whooping-cough among the infectious diseases, in the ordinary sense of the term, urge us to separate mumps from them also, in spite of its contagiousness. Infants and old persons usually escape epidemic parotitis; males are more frequently attacked than females.

*Symptomatic parotitis* results from severe diseases, like typhus; in some epidemics of this disease it follows almost all cases. More rarely it is seen in the course of cholera, septicæmia, measles, small-pox, dysentery, or as an accompaniment of pneumonia. We do not exactly know the relation of such cases of parotitis to these diseases. The oral catarrh, which always accompanies abdominal typhus, might excite the suspicion that the parotitis accompanying this disease was induced by a propagation of the catarrh of the mucous membrane of the mouth along the excretory ducts of the glands. But, opposed to this view is the fact, that the frequency with which it occurs in typhus is not proportionate to the intensity of the affection of the oral mucous membrane, as well as the circumstance that parotitis, running the same course, occurs in other affections that are not complicated by oral catarrh. Since symptomatic parotitis is seen not only in infectious diseases, but also in pneumonia, we cannot say that it is induced by an irritation of the gland from infected blood. The hypothesis, that under some circumstances it has a critical indication, and exercises a favorable influence on the course of the original disease, is disproved by facts; it always forms an unpleasant and undesirable complication.

Anatomical Appearances.—We do not exactly know the ultimate anatomical changes of parotitis. As the course of the disease is almost always favorable, there is rarely an opportunity for anatomical examinations. Nevertheless, from the softness of the swelling, and the slight amount of pain that it causes, and especially from its usually rapid disappearance, without leaving any traces, we may believe that it is chiefly or solely caused by serous exudation. Although we have said above that the affection probably proceeds from a catarrh of the gland-duct, still there is no doubt that the swelling chiefly affects the interstitial substance and the connective tissue about the gland. The swelling usually extends far beyond the borders of the gland. The development of œdema about the inflamed gland-ducts is not at all strange; it corresponds exactly with observations made in analogous conditions. Infiltration, with firm fibrinous exudations and suppuration, rarely occur in parotitis. We do not know whether the suppuration proceeds from the gland itself or the interstitial substance; but it is most probable that there is just the same state of affairs as in suppurating symptomatic parotitis. (See below.)

*Symptomatic parotitis* begins, according to the careful observations of *Virchow*, with decided hyperæmia, which causes the gland and interstitial substance to appear infiltrated and swelled. Changes in the gland-ducts soon begin; a tough, filamentous, whitish, or yellowish substance, which soon becomes purulent, collects in them. Even at the second or third day the microscope shows that it contains pus-corpuscles, with numerous salivary corpuscles. If the disease proceeds, the lobules of the gland soften and break down; this process begins within, so that at one time the lobules represent cavities filled with pus. Finally, the tunica propria is also destroyed, and the interstitial tissue begins to suppurate; this suppuration may extend rapidly, and become a diffuse phlegmonous inflammation. In this case a large parotid abscess forms; more frequently the gland-tissue only is destroyed, and as the interstitial tissue remains intact, numerous small abscesses are formed. Occasionally also there are extensive destruction and gangrene of the gland-tissue and interstitial substance; the inflammation and suppuration may spread from its original seat in various directions and cause dangerous results. It most frequently attacks the neighboring connective tissue, and the masticatory muscles lying in it, the periosteum of the maxillary, temporal, and sphenoid bones, or even the bones themselves. Where the disease is very severe, it occasionally passes from the bones to the membranes of the brain, and the brain itself, or to the internal and middle ear. This propagation of inflammation and suppuration to the cerebral membranes and the internal ear may take place along the blood-vessels and nerve-sheaths, as well as through the bones. Finally, in some cases parotitis induces phlebitis and thrombus of neighboring veins, especially of the anterior and posterior facial and external jugular veins; the disintegration of these thrombi may cause embolism and septicæmia.

SYMPTOMS AND COURSE.—In idiopathic parotitis, as in other inflammations, the local symptoms are often preceded by slight fever.

The general disturbance, depression, headache, loss of appetite, restless sleep, etc., accompanying this as other fevers, are usually called the premonitory symptoms of idiopathic parotitis. After the fever has lasted two or three days, or in some cases simultaneously with its occurrence, a swelling forms, which, beginning near the lobe of the ear, rapidly extends over the cheek and to the neck; usually only one side is at first affected. In the middle it is firmer, at the periphery softer; the skin over it is pale or only slightly reddened. This swelling is accompanied by a feeling of tension and pressure, but by no severe pain; the motions of the head are impaired, the mouth can only be slightly opened, and chewing and swallowing are difficult. The secretion of saliva may be increased, diminished, or unaltered. These annoyances are so slight in proportion to the disfigurement which gives the name to the disease, that the patients excite more laughter than sympathy. The swelling almost always soon extends to the other side of the face, and is often greatest there when it has gone from the side first affected and the fever has subsided. About the fifth or sixth day, occasionally even earlier, rarely later, the fever ceases, and after eight or ten days the face appears natural. But sometimes a circumscribed, painless, hard swelling remains for a while in the region of the parotid. Far more rarely about the fifth or sixth day the swelling becomes very painful, hard, dark red, and abscesses form, which open outwardly or into the external auditory meatus.

Occasionally, in the course of the disease, one of the testicles is affected by an inflammation similar to that of the parotid; this occurs more frequently in men than in boys; it is usually accompanied by pain in the sacral and inguinal regions and exacerbation of fever. The scrotum also becomes œdematous and forms an inelastic, doughy tumor, which is not often reddened; on careful examination we readily find that there is a serous exudation in the tunica vaginalis. Inflammation of this part usually runs as favorable a course as that of the parotid does, and after a few days terminates in resolution. Occasionally the parotitis and orchitis seem to alternate, as it were; the former disappears as the latter comes on, and the reverse: hence we speak of parotitis polymorpha being "fugitive," and of its inclination to metastasis to the testicle. In other cases, however, the two inflammations run on together, which renders it probable that both are due to the same cause, and that the occurrence of the one is not to be regarded as due to the disappearance of the other. As in men the scrotum is sometimes affected, so in women the vulva or breasts are occasionally attacked with inflammatory œdema. In other cases, pain in the region of the ovaries, increased by pressure, shows that an ovary is inflamed, just as the testicles are in men. Cases have also been recorded, where, in the course of idiopathic parotitis, fatal meningitis has been developed.

When *symptomatic parotitis* occurs at the height of typhus or any of the above-mentioned diseases, the apathetic patients do not usually complain of pain or any other symptom. Occasionally slight chills or an exacerbation of fever precede the formation of the parotid tumor. This sometimes forms gradually, at others very rapidly, and generally affects only one side. If parotitis comes on during convalescence from typhus, etc., it is accompanied by the same symptoms that we have described for idiopathic parotitis. Symptomatic parotitis also may end in resolution. This occurs most readily when the tumor has formed gradually and attained only a moderate hardness and extent. The diminution in size is sometimes slow, sometimes rapid. When about to suppurate, the swelling becomes uneven, nodulated, and very red; it usually shows fluctuation at several points, and, when opened spontaneously or artificially, benign pus is evacuated. Occasionally the opening occurs simultaneously outward, and into the external auditory meatus, more rarely into the mouth or pharynx. Finally the pus may burrow along the sterno-cleido-mastoid muscle, or the œsophagus and trachea, and form abscesses at the lower part of the neck or even enter the chest (*Bruns*). While mortifying, the skin covering the tumor becomes dark blue and discolored; the tumor, which was previously hard, becomes doughy and sinks in; after a spontaneous or artificial opening, a discolored pus, mixed with shreds of tissue, is evacuated.

TREATMENT.—As idiopathic parotitis almost always ends in a cure, if left to itself, we have little to do but protect the patient from injurious influences, and to regulate the digestion and bowels, while the disease lasts. We keep the patient in his chamber, cover the swelling with wadding or a spice-bag, and as long as the fever lasts let him avoid eating much meat or other proteïn substances which would not be readily digested (see diseases of the stomach). In some cases an emetic or laxative may be necessary. If hardness and greater sensibility of the swelling, with increase of the fever, excite fears of suppuration, we may attempt to check it by applying leeches. If we find fluctuation, we should apply cataplasms, and open the abscess early, to prevent further destruction of the parotid, or perforation of the pus into the external auditory meatus. Irritant applications have been used to prevent metastases, and sinapisms and blisters have even been applied to the parotid region, to induce a return thither of the inflammation that had affected the scrotum and testicles. As experience has shown, such treatment can only prove injurious.

In symptomatic parotitis even local blood-letting is badly borne, on account of the severity of the original disease. If the swelling be red, and the patient winces when we press on the tumor, we should apply compresses of cold water or ice. When there is fluctuation, warm poultices and early opening of the abscess are indicated.

## CHAPTER XI.

### SALIVATION—PTYALISM.

STRICTLY speaking, we have no right to consider salivation as a distinct disease (it forms a symptom of a great variety of affections); but we follow custom in giving a separate chapter to the anomalies of secretion of the salivary glands. The quantity of saliva secreted in twenty-four hours is usually estimated at ten to twelve ounces, but it varies considerably even during health. It is best, with *Wünderlich*, to consider the increased secretion as *disease* or *salivation*, when it ceases to pass into the stomach with the ingesta; but some of it flows out of the mouth, some is spit out, or is swallowed by itself, because it becomes troublesome.

ETIOLOGY.—Physiology sufficiently explains the causes of most forms of salivation; in other cases we do not know them.

Salivation is caused—1. By irritation of the mucous membrane of the mouth or pharynx. Introduction of irritating substances into the mouth excites the flow of saliva, which consequently occurs in most of the affections of the mouth described in the preceding chapters, as well as in almost all surgical affections of the mouth. According to the beautiful experiments of *Ludwig*, the flow of saliva is increased by irritation of certain nerves, such as the lingual branch of the trifacial or the glossopharyngeal; this increase also occurs when these nerves are divided and their central ends irritated. Of course the irritation of the divided nerves must be transferred to the nerve filaments governing the secretion of saliva, which is then to be regarded as a reflex symptom. In the same way we may regard the flow of saliva caused by the irritation of the peripheral expansions of the glossopharyngeal and lingual nerves induced by acrid ingesta, by wounds or ulcers, as a reflex symptom. Probably the salivation observed in neuralgias of the trifacial results fromt he same cause. The increase of saliva due to the use of mercurial and iodine preparations appears to depend not on the simple addition of these substances to the secretion, but to the irritation of the mouth, produced by excreting them for a long time. For they must be long taken before the secretion of saliva is greatly increased; salivation does not begin till the mouth becomes diseased from their continued action. Corresponding to this, *Lehmann* found that at the commencement of mercurial ptyalism the excretion was not saliva but mucus, mingled with shreds of epithelium from the oral mucous membrane. The preparations of iodine, which induce stomatitis less frequently, cause salivation far more rarely, although we can detect their presence in the saliva quite early. We do not know whether the salivation produced by muriate of gold and other metallic and vegetable substances originates in the same way.

2. In many cases, salivation appears to depend on irritation, affecting the gastric or intestinal mucous membrane, perhaps also the uterus or other organs. *Frerichs* has shown, by experiment, that irritation of the gastric mucous membrane increases the secretion of saliva; for, when he introduced food into the stomachs of dogs through fistulous openings, there was profuse salivation; if he used common salt, quantities of saliva flowed from the mouth. These experiments appear to prove that irritation of the gastric nerves is also reflected to the nerves governing salivation, and they at least partly explain the increased flow of saliva accompanying many pathological states of the stomach, such as ulcer or cancer of the stomach, and preceding vomiting, whether induced by emetics, overloading, or disease of the stomach. It seems probable, also, that the same cause induces the salivation so constantly accompanying the pains produced by worms in the intestines, that the laity who are aware of this symptom have the most wonderful hypotheses about the flow of water into the mouth from the irritation of worms. We have less reason for referring the salivation which not unfrequently occurs during the first months of pregnancy, or in hysteria, to an excitement of the genital nerves reflected to the secretory nerves of the salivary glands.

3. Salivation depends on certain mental influences. We see how the secretion is increased in disgust or desire. As a proof that abnormal excitement of the brain may directly increase the secretion of saliva, we may note the fact that physiologists have been obliged to locate the origin of the nerves, governing the secretion of saliva, in the brain. The activity of the salivary glands is increased in the same way by irritation of the trigeminus and facial nerves, even at points where no sympathetic filaments are mingled with them, that is, above the ganglia.

4. Occasionally salivation occurs in the course of diseases, such as typhus, intermittent, etc., without other perceptible cause; its occurrence in these diseases has even been regarded as critical.

Finally, some apparently healthy persons suffer from obstinate salivation without perceptible cause. In insane and old people, the flow of saliva from the mouth does not appear to depend on its increased secretion, but on neglecting to swallow that which is produced in normal amount.

ANATOMICAL APPEARANCES.—We do not know the anatomical changes undergone by the salivary glands in increased salivation. In continued and excessive salivation, slight swelling of the parotid occurs in some rare cases. The fact, that the secretion may still be obtained after the heart has ceased to beat, proves that overloading of the vessels, or hyperæmia of the salivary glands, which instantly causes their infiltration and swelling, is not the sole cause inducing increased secretion.

SYMPTOMS AND COURSE.—The pains in the mouth, and painful swellings of the neighboring lymphatics, which occur in salivation, belong to the various forms of stomatitis exciting it; salivation itself causes no pain, but it greatly inconveniences the patient. The frequent collection of fluid in the mouth obliges him to spit constantly; frequently he cannot speak two words without interruption. Rest at night is also disturbed, partly by the saliva flowing from the mouth and wetting the pillow, partly by that which, flowing backward, passes into the pharynx and larynx. The escaping fluid may reach the amount of six or eight pounds in twenty-four hours. *Lehmann* and other observers have found it, at first, more mucous, cloudy, of greater specific gravity, and richer in solid constituents (young and old epithelial cells), than normal saliva. The fluid is alkaline, contains much fat and little ptyaline, and only rarely perceptible amounts of sulpho-cyanide of potassium. Subsequently, the secretion was less cloudy, and, like the saliva that *Ludwig* obtained, by continued irritation of nerves influencing the secretion, it contained less solid constituents than normal saliva. This fluid was also alkaline, rich in fat and so-called mucous corpuscles; it contained no sulpho-cyanide of potassium. When salivation has continued a long while, albumen may occasionally be found in the fluid. The patients usually emaciate; the loss of water and organic constituents has little to do with this, but, as the accompanying stomatitis interferes with chewing, the patients take little nourishment, and what they do take is badly assimilated, because the quantity of saliva swallowed interferes with digestion.

TREATMENT.—The causal indications require a careful treatment of the original disease when the salivation is caused by affections of the mouth. When resulting from the misuse of mercurials, slight laxatives are to be recommended. *Cullerier* calls constipation "one of the best known of the exciting causes of salivation," and, indeed, it is more rational to suppose that the mercurials which reach the mouth through the salivary glands, and are swallowed, would be more readily removed by purgatives than by remedies directed to the skin or kidneys. Salivation caused by affections of the stomach, intestines, uterus, etc., is also most readily improved by proper treatment of the original affection; in other forms the causal indications cannot be fulfilled.

For the indications of the disease, derivatives, "general baths, application of blisters and mustard to the throat and nape of the neck," astringent mouth-washes of alum, sulphate of zinc, sage, or oak-bark, have been recommended. The use of opium deserves most confidence. It is always satisfactory when, as in this case, theory and practice agree in supporting a therapeutic measure. The use of opium in salivation was recommended by the first practitioners of medicine; and, since salivation depends on excitement of the nerves, it appears rational to use for it remedies which, like the narcotics, diminish the excitability of the nerves. There are cases of spontaneous salivation that defy all treatment.

# SECTION II.

## *AFFECTIONS OF THE PHARYNX.*

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## CHAPTER I.

### CATARRHAL INFLAMMATION OF THE PHARYNGEAL MUCOUS MEMBRANE—ANGINA CATARRHALIS.

ETIOLOGY.—The disturbances of function and nutrition, which we have frequently designated as characteristic of catarrhal inflammation, are often observed in the mucous membrane of the pharynx, the soft palate, the uvula, and tonsils, and are usually termed *angina catarrhalis.* In this section, the tissues of the soft palate are considered as belonging to the pharynx, since they participate in almost all the diseases of the pharynx.

The predisposition for catarrhal inflammation of the pharynx varies with the individual. If exposed to the slightest injurious influences, some persons are immediately attacked with affections of this part, while others, exposed to the same influences, remain well, or have disease of some other part. Some persons are troubled several times a year with catarrhal angina, while others live for years without having it. The causes of the increased predisposition to catarrhal angina are mostly unknown. It is customary to say that a lymphatic constitution predisposes to the disease, or that it is more apt to occur in scrofulous persons. But we often see robust individuals, who show no constitutional anomalies, affected with catarrhal angina at every exposure. In general, we may say that the disease is more common in children and young persons than in those more advanced in life; that repeated attacks leave an increased predisposition; that patients who have had syphilis, or who have used mercurials for a long time, are peculiarly liable to acute and chronic pharyngeal catarrh.

Among the *exciting causes* are: 1. Direct irritation; such as hot or corrosive substances, rough, ragged bones, which stick in the fauces, and other injuries to the pharyngeal mucous membrane. Perhaps the catarrh induced by spirituous liquors depends on their direct action. 2. In other cases, the catarrh undoubtedly depends on *catching cold*. 3. Not unfrequently it is propagated from neighboring parts to the pharyngeal mucous membrane. In this class belong the catarrhs occurring in mercurial stomatitis, and those difficulties of swallowing which accompany the later stages of laryngeal catarrh. Sometimes it accompanies catarrh of the stomach; but every angina is not, as was formerly supposed, of gastric origin. Not unfrequently catarrhal angina must be regarded as the result of a blood-disorder. It is not a complication, but a symptom of scarlatina, which is just as constant as the exanthema. More rarely in exanthematous typhus, or measles, which are always accompanied by catarrh of the respiratory organs, there is also pharyngeal catarrh. Among the chronic infectious diseases, constitutional syphilis often makes its appearance as pharyngeal catarrh; but other changes in the tissues of the pharynx usually occur soon, which will be spoken of hereafter. 5. Sometimes catarrhal angina is epidemic. A large number of persons are taken sick without our knowing the influences inducing the affection. In many other cases, also, the exciting causes are unknown.

ANATOMICAL APPEARANCES.—In acute catarrhal angina, the mucous membrane, especially that of the soft palate, appears dark red. The swelling of the mucous and sub-mucous tissue is most evident at the uvula, which has plenty of relaxed sub-mucous tissue. The uvula is thicker, but especially longer, and often rests on the root of the tongue ("the palate is down"). The tonsils also are more or less swollen. At first the mucous membrane is dry; later, it is covered with cloudy secretion, particularly about the tonsils and posterior wall of the pharynx.

In *chronic* catarrh of the fauces, the membrane does not appear regularly reddened; it is traversed by varicose veins, and is darker-colored. The swelling is greater and more irregular than in the preceding variety. The diseased mucous membrane sometimes appears dry and glistening, sometimes covered with a cloudy secretion. On the soft palate and uvula the swollen and closed glands often appear as small granules, or they form small yellow vesicles, which soon rupture, and leave round (follicular) ulcers. In the dilated openings of the tonsils there are occasionally found cheesy, badly-smelling plugs or stony concretions, which are the putrefied or petrified contents of the follicles.

Recently, chronic pharyngeal catarrh has received a great deal of attention in the journals and treatises on baths, but it has not been sufficiently considered in the text-books on pathology. Occasionally it is limited to the pharyngeal mucous membrane; again, it extends to that of the larynx or nares. The changes consist in an irregular hyperæmia, so that sometimes we see only a few varicose vessels in the otherwise pale membrane; in a thickening, or hypertrophy, which is either diffuse, or limited to undefined spots; and, in a perverted secretion of the mucous membrane. From the partial thickening of the mucous membrane, in which the sub-mucous tissue also participates, the posterior wall of the pharynx acquires a peculiar nodulated appearance; there are numerous round, or oval, sometimes confluent prominences, whence the disease is called *pharyngitis granulosa.* Some authors designate it pharyngitis follicularis, because they consider that the partial hypertrophy of the mucous membrane is chiefly limited to the vicinity of diseased mucous glands. This view is probably correct, but has not yet been anatomically proved. In some cases the secretion of the mucous glands is very abundant, and then it sometimes shows an inclination to dry into disgusting yellow or green crusts; in other cases it is scanty, and then also shows the inclination to dry, and the posterior wall of the pharynx looks as if covered with a thin coat of varnish. For this form of the affection, *Lewin* has proposed the very suitable name of pharyngitis sicca.

SYMPTOMS AND COURSE.—Acute catarrhal angina is usually accompanied by a fever, which has the symptoms of catarrhal fever, as previously described; this occasionally precedes the local difficulties, but it is sometimes, though rarely, entirely absent. At first the secretion from the mucous membrane is diminished; hence the patients complain of dryness in the throat. From the tension of the mucous membrane, especially at the half arches of the palate, where it is closely attached to the subjacent muscles by a scanty connective tissue, there is great pain, which is so increased at every attempt to swallow that the patients make wry faces whenever they attempt it. When, as frequently happens, the elongated uvula touches the tongue, there is a sensation of a foreign body in the throat and a constant inclination to swallow. In very severe forms of catarrhal angina, which are often called erysipelatous or erythematous angina, the muscles of the palate are often infiltrated with serum and their functions limited. Under normal circumstances, as is well known, the contraction of the muscles of the anterior half arches of the palate prevents the return of food into the mouth; contraction of the muscles of the posterior half arches closes the passage to the nose, as the uvula fills up the opening that is left. If the function of these muscles be impaired, fluids would be driven through the nose or back into the mouth by the contractions of the pharynx in the attempt to swallow. If the mucous membrane of the *pharynx* be the seat of an intense catarrh, and, as a consequence, the muscles be paralyzed by serous infiltration, the patient suffers still more. As soon as a morsel of food, or, still more, any liquid, has passed the anterior half arches, the patient is greatly terrified, as he cannot pass it either forward or backward. As the substance in the pharynx would pass into the larynx on any attempt to breathe, the patients hold their breath and attempt, in every conceivable manner, to evacuate the contents of the pharynx through the mouth; they bend far forward and let the head hang over the side of the bed. Nevertheless, some of the contents of the pharynx often enter the larynx, and are again expelled by spasmodic coughing. The patients at last become timid, and, with terror, wave back the drink or medicine offered to them, they pass day and night in the most uncomfortable postures, so that the saliva may flow out of the mouth, and they may not be obliged to swallow it. A "nasal" tone of the voice is a pathognostic symptom of all affections of the pharynx, where the functions of the muscles of the half arches of the palate are affected, and consequently for all the intense forms of catarrhal angina. As is well known, it is only in saying *N* and *M* that we allow the air to pass through the nose; while pronouncing other letters, the nasal cavities are closed. When patients are unable to shut off the nose in this way, from inability to contract their posterior half arches, the resonance of the nose gives to all sounds a peculiar tone, which is called "nasal," and the person is said to "speak through the nose." Besides this difference of tone, there is a certain difficulty of speech. The patients speak slower and more carefully, because it pains them, especially when saying *N*, in doing which the root of the tongue is for a moment pressed against the roof of the mouth. A last characteristic is, that the pronunciation of guttural *R*, in which the uvula is made to vibrate, becomes difficult or even impossible, if the uvula is much swollen and elongated.

As we said in the first chapter of the previous section, the milder as well as the more severe forms of catarrhal angina are almost always accompanied by catarrhal stomatitis. The patients have a coated tongue, bad taste, foul breath, and the mouth is always full of saliva. Not unfrequently acute pharyngeal catarrh extends to the Eustachian tubes and the tympanum; the patients become deaf, have piercing pains in the ears, which may be excessive, until perforation of the drum permits the escape of pus from the middle ear, when a remission suddenly occurs.

Catarrhal angina almost always terminates in recovery after a few days. While the pain and difficulty in swallowing subside, quantities of mucus are removed from the pharynx by hawking and spitting; at the same time the symptoms of oral catarrh pass away.

In *chronic* catarrh of the fauces, the pain and difficulty of swallowing are usually slight, and only become worse occasionally when the chronic catarrh is exacerbated by slight injuries. This is particularly true of the chronic catarrh of the soft palate, which is very frequent in patients who have suffered from syphilis, or who have used mercurials for a long while. The slight difficulties that these patients experience in swallowing, and the temporary exacerbations which occur, are a source of unceasing care. They usually soon attain great skill in looking at their own throats in the mirror; the smallest phlyctenula coming on the soft palate does not escape their notice; they constantly run after the doctor, who must again look in their mouth, and again assure them that they are not syphilitic. The cheesy plugs which form in the tonsils are occasionally ejected by hawking. This symptom also troubles the patient a great deal; the yellow, round bodies, which smell horribly when squeezed, are to them a sure sign that they have tubercles; and it is as difficult to convince the latter patients that they are not consumptive as to satisfy the former that they are not syphilitic. Chalky concretions from the tonsils, which are hawked up, are usually represented as lung-stones. The milder cases of chronic pharyngeal catarrh, from which most habitual drinkers suffer, usually trouble the patients only in the morning, when the mucous membrane secretes most abundantly a tough mucus, or when it is covered with the mucus secreted during the night. The patients attempt to remove this secretion by continued hawking and spitting, and this straining, which not unfrequently causes nausea and vomiting, is one of the causes of the notorious morning vomiting of drunkards. The severe forms of chronic pharyngeal catarrh, especially of the follicular or granular varieties, are far more troublesome. They do not render swallowing actually difficult, but the patients complain of an irritation, a disagreeable sensation of prickling, also of dryness in the throat, which leads them unwillingly to make the motion of swallowing, or, more frequently, to hawk and hack for a long while. It is thought, too, that this repeated hawking is a bad habit. The voice often becomes husky also, as the laryngeal mucous membrane usually participates in the affection. When the follicular pharyngeal and laryngeal catarrh is exacerbated, the hacking increases to a troublesome spasmodic cough, and the husky voice becomes actually hoarse. If the affection extends to the nasal mucous membrane, the nose becomes stopped at night; hence the patients sleep with the mouth open, and by morning the pharynx and back of the tongue have become so dry that moving them causes cracks in the dry coating, or even in the membrane itself, and then there are slight hæmorrhages. Many patients are greatly worried by this spitting of blood, whose origin can hardly be discovered, unless we see the patient just after he has awakened; and thus follicular catarrh of the pharynx and larynx, which is a very obstinate, although not dangerous disease, has a very depressing effect on most patients.

Treatment.—When of moderate intensity, acute catarrhal angina does not require any particular treatment. Often the patients do not apply to a physician, but go to some old woman, who knows how to raise the "fallen palate" by certain hairs at the top of the head. These foolish ideas have a serious as well as a ridiculous side. The apparent success of this and similar senseless procedures must teach us to abstain from energetic treatment in affections where they have a great reputation. This teaching is much opposed in the treatment of catarrhal angina. We might say that more than half the physicians superfluously give an emetic, partly with the idea that it will act as a revulsive, partly to combat the gastric disorder, which is diagnosticated from the symptoms of oral catarrh, on which the angina is thought to depend. As the tongue is cleaner the day after the emetic, and the angina has improved, as it would have done at any rate, the remedy receives the credit of it.

In catarrhal angina, the use of an emetic is only admissible under certain circumstances, as when there are substances in the stomach that have excited, or are keeping up, a gastric catarrh. In severe cases it is well to let the patient apply moist compresses, well wrung out, and carefully covered with a dry cloth, to the throat, every few minutes. In persons who are afraid of the cold compresses, or where, for any reason, we do not wish to use these, we may employ warm poultices. At the same time, we may have the mouth frequently washed with cold water, or with a solution of alum, sulphate of zinc, acetate of lead, etc. Occasionally, by covering the inflamed spots with powdered alum, or painting them with a solution of nitrate of silver, ʒ j to ℥ j, we may abort the disease.

*Chronic catarrh of the fauces* is best treated by the above-named astringent mouth-washes, and particularly by painting the inflamed spots with solution of nitrate of silver.

*Chronic pharyngeal catarrh* must be very carefully and continuously treated; in many cases it defies medical skill. In the blennorrhœal form even, which offers the best prognosis, treatment often fails, because the patients cannot decide to give up the use of liquor, or to smoke less. The best treatment in these cases is the local application of solutions of nitrate of silver, alum, or tannin, and these seem to be more efficacious when given in a nebulized form than when applied with a brush. In the forms where there is little secretion, and particularly in the follicular and granular pharyngeal catarrh, it occasionally appears as if the application of the above solutions caused a "toning up" of the affected mucous membrane, and an improvement of the disease, but in the cases that I have seen, this improvement has only been apparent, or, at least, only temporary. Solutions of corrosive sublimate or sulphuret of lime have not proved more beneficial. Recently, as recommended by *Lewin*, I have, in some cases, tried a *Lugol's* solution (℞ iodin. gr. vj; potass. iodid. gr. xij; aquæ ℥ vj), for painting the pharyngeal mucous membrane, and, although I have not used it in a great many cases, it seems preferable to other remedies in dry catarrh of the pharynx, with or without granulations. In this form of chronic pharyngeal catarrh the alkaline muriatic mineral waters have the best reputation, particularly those of Ems and the sulphur springs, especially those of Weilbach, and some Pyrenean springs.

## CHAPTER II.

### CROUPOUS INFLAMMATION OF THE PHARYNGEAL MUCOUS MEMBRANE —PHARYNGEAL CROUP.

**Etiology.**—In the croupous inflammation of the pharyngeal mucous membrane, the croup membrane often adheres so firmly to the inflamed mucous membrane that, on detaching it, a bloody, superficial loss of substance remains. Then the affection shows a change from croupous to diphtheritic inflammation.

1. Pharyngeal croup occurs as an independent disease, from the same causes as pharyngeal catarrh, and it almost seems as if it were occasionally only a more intense form of catarrh. 2. The croupous deposits on the tonsils, so often seen in parenchymatous angina, are explained by the intense participation of the mucous membrane in the inflammation of the subjacent tissues. 3. Pharyngeal croup, which appears as a symptom of a sporadic, or, more frequently, epidemic croupous inflammation, affecting the mucous membrane of the palate, pharynx, larynx, and trachea, is very important. In this form the croup sometimes seems to spread from the larynx to the pharynx (croup ascendant), sometimes the reverse (croup descendant). 4. Lastly, pharyngeal croup occurs with croupous and diphtheritic inflammations of other mucous membranes in the later stages of typhus, in septicæmia, and similar diseases, a form which we shall not consider further at present.

**Anatomical Appearances.**—We see white or grayish-white membranous masses on the reddened mucous membrane of the soft palate, tonsils, and pharynx. They usually form small, irregular, roundish islands; more rarely extensive membranes. Under these there is no loss of substance.

**Symptoms and Course.**—Idiopathic, uncomplicated croupous angina causes the same annoyances as severe catarrhal angina; we only discover the form of the inflammation by inspecting the pharynx. On careless examination, the gray patches may be mistaken for ulcers, with fatty bases.

The subjective symptoms of parenchymatous angina are not altered by croup, so that, in this case also, the croup is first recognized on inspecting the pharynx.

Croupous angina, which usually occurs epidemically with croupous laryngitis, is easily overlooked, as it causes proportionately little difficulty, which, moreover, will probably be misunderstood, as it affects children almost exclusively. If we examine the fauces of children sick with croup, we often find them covered with croup membrane, although the parents may not have noticed that the children had any difficulty in swallowing. We have before said how important for diagnosis and prognosis it is to examine the throat of every child affected with hoarseness.

TREATMENT.—The treatment of croup occurring idiopathically, after catching cold, etc., is the same as that for the severe forms of pharyngeal catarrh.

Pharyngeal croup accompanying croupous laryngitis requires, as we have already said, the prompt removal of the membrane, and energetic cauterization of the affected mucous membrane, with a concentrated solution of nitrate of silver.

## CHAPTER III.

### DIPHTHERITIC INFLAMMATION OF THE PHARYNGEAL MUCOUS MEMBRANE.

DIPHTHERITIC inflammation, in which a fibrinous exudation is deposited in the tissue of the mucous membrane, and presses on its vessels so as to cause it to slough, attacks the pharynx very frequently. Diphtheritic pharyngitis, however, does not occur as a primary and independent affection, but in almost all cases depends on infection of the blood from the poison of scarlatina, or of the disease we call epidemic diphtheria and class among the infectious diseases. (We speak of croupous and diphtheritic inflammations of the different mucous membranes; but when we speak of "croup," or "diphtheria," we always mean croupous inflammation of the laryngeal mucous membrane or diphtheritic inflammation of the pharyngeal mucous membrane.) We shall hereafter give a detailed account of diphtheria, when speaking of scarlatina and epidemic diphtheria.

## CHAPTER IV.

### PHLEGMONOUS INFLAMMATION OF THE PHARYNX.

Etiology.—The submucous tissue of the pharynx and the interstitial tissue of the tonsils, which are the seat of simple œdema in catarrhal and croupous inflammation, may also suffer from inflammatory disturbances of nutrition. These often consist in infiltration of the tissue with fibrinous exudation, and in proliferation of the connective tissue; in other cases pus is formed, the tissues melt away, and abscesses result; diffuse mortification and phagedæna of the affected parts occur in some rare cases.

The same causes, according to their intensity, or the predisposition of the patient, appear capable of exciting the catarrhal and parenchymatous forms of pharyngeal inflammation; hence we refer to the etiology of the catarrhal form. Parenchymatous pharyngitis also leaves great tendency to relapse; the more frequently it has affected a person, the more liable he is to have it again. Many persons have it yearly, or even oftener. Once having ended in suppuration, it seems disposed to take the same course on subsequent occasions, so that, in such cases, in new attacks, there is little hope of causing the disease to end in resolution.

Anatomical Appearances.—Acute parenchymatous pharyngitis usually attacks the tonsils; one or both may be inflamed, sometimes the inflammation passes from one to the other. From the exudation, with which they are infiltrated, the tonsils often swell to the size of a walnut; their surface appears nodulated, dark red, covered with glutinous exudation or croupous deposits. As the inflammation passes on to suppuration, some circumscribed spot usually becomes softer and more prominent, and finally the pus perforates the thinned walls of the abscess. More rarely the acute parenchymatous inflammation occurs in the submucous tissue of the soft palate; a hard swelling forms here, and fluctuation gradually occurs; finally, in this case also, the pus is evacuated into the mouth or pharynx.

*Chronic* parenchymatous pharyngitis also almost exclusively affects the tonsils; more rarely the uvula, or the soft palate, is permanently thickened by inflammatory hypertrophy of the submucous connective tissue. From this cause the tonsils may become very large and hard; their surface is often uneven and nodulated, and has depressions where there was formerly a loss of substance from suppuration. The mucous membrane is but slightly reddened, or is even pale. We often find the above-described cheesy plugs in the gaping openings on the surface of the tonsils.

SYMPTOMS AND COURSE.—Acute parenchymatous pharyngitis generally begins with a high fever, which may be preceded by a severe chill. The general condition of the patient is much affected, the pulse full and frequent, the temperature 104°, or over. In this case we have not, as in pharyngeal catarrh, a catarrhal, but we have an *inflammatory* fever, such as accompanies pneumonia and other inflammations of important organs. It is only in rare cases, where the disease is not severe and runs a very sluggish course, that the fever is moderate. With the commencement of the fever, or, perhaps, not till next day, the patients complain of a feeling of tension and soreness in the throat, and often of piercing pain, extending toward the ear; it feels to them as if there were a foreign body in the pharynx, hence they make constant attempts to swallow, although the motion increases their pain. Sometimes all the painful and terrifying symptoms occur, which we described in the first chapter of this section as accompanying the severer forms of catarrhal pharyngitis. Not only does swallowing become very painful, so that, when the patient attempts to swallow a little saliva, he distorts the face; but, from the imbibition and paralysis of the muscles of the palate and pharynx, when he attempts to swallow, both solids and fluids come back through the mouth and nose, or else we have the painful and dangerous condition that we have before described (p. 448) as caused by the impossibility of getting the morsel out of the pharynx. The secretion of saliva is often enormously increased; if the patient opens the mouth, without spitting, the saliva runs from the corners of the mouth. The tongue is thickly coated, the odor from the mouth very unpleasant; there is also the characteristic modification of the voice; its resonance is changed, the speech has the peculiar nasal twang, from which alone we may often suspect the disease as soon as the patient speaks. Other characteristics of parenchymatous angina are the difficulty and pain caused by opening the mouth; frequently the patient cannot separate the teeth more than a few lines; this difficulty is apparently caused by the excessive tension of the bucco-pharyngeal fascia. Respiration is affected far less frequently than speech and the opening of the mouth. Any considerable want of breath, added to the symptoms of parenchymatous angina, is always a serious symptom, and must arouse the suspicion that there is œdema glottidis. On examining the mouth and pharynx, which is done with difficulty, we often find the tonsils so swollen as to touch each other or to squeeze the œdematous uvula between them. If only one tonsil be inflamed, we often see the uvula pressed entirely to the opposite side. We find the soft palate pressed forward into the middle of the mouth. At the part of the neck corresponding to the tonsil, that is, behind and below the angle of the lower jaw, we find a hard, painful swelling. Even more frequently than in catarrhal pharyngitis, the inflammation extends, with severe pain, to the Eustachian tube and tympanum. While the local symptoms thus increase for three or four days, the fever grows higher, and symptoms of hyperæmia of the brain occur; the patient has severe headache, is sleepless, tormented by horrible dreams, or even becomes delirious. When the inflammation ends in resolution, the local and general symptoms usually subside toward the end of the week, and the patient generally recovers in eight to fourteen days. When suppuration occurs, and abscesses form, there is a sudden remission after the symptoms have reached their highest point. The patients often perceive the opening of the abscess only by the sudden relief they experience, as the pus may be swallowed or overlooked; in other cases the opening may be instantly recognized by the fetid odor and the yellow color of the substance thrown out. It is doubtful how the pus, which has been completely enclosed and protected from the air, acquires this very disagreeable smell. After the opening of the abscess convalescence is generally rapid.

Acute parenchymatous inflammation of the soft palate gives subjective symptoms similar to those of acute tonsillitis, and we can only decide on the presence of one or the other by the objective appearances.

*Chronic* parenchymatous angina either results from protracted attacks of the acute form, or comes on gradually and independently. It generally causes very little trouble; there is little or no pain, the increased mucus is due to the accompanying catarrh; but the slightest irritation causes the chronic to relapse into the acute form again. The speech is often changed by the hypertrophy of the tonsils; in other cases pressure on the Eustachian tubes causes permanent deafness. The enlarged and elongated uvula may irritate the entrance to the glottis, and so excite habitual spasmodic cough.

TREATMENT.—General and local blood-letting are recommended in acute parenchymatous angina. The former, which *Bouillaud* employed as "saignées coup sur coup," is never required by the disease itself, and but rarely by its complications. Leeches, applied to the neck, give little ease, and even scarification of the tonsils has not done as much good as was expected of it.

If called the first or second day of the disease, we may employ the treatment advised by *Velpeau;* that is, apply powdered alum to the inflamed part two or three times daily, and advise the patient to rinse his mouth frequently with a solution of alum (ʒ iij—℥ ss to ℥ vj of barley-water). Instead of alum, solid nitrate of silver has been recommended to cut short the disease.

If called in later, or if the *Velpeau* treatment has been unsuccessful, the energetic use of cold is a rational treatment, whose benefit is proved by experience. We let the patient take ice and cold water in the mouth, and cover the throat with cold compresses, which must be frequently renewed.

If fluctuation occurs, we should apply warm poultices to the throat, wash out the mouth frequently with camomile-tea, and open the abscess early with the finger nail, or with a bistoury, covered to near the point with adhesive plaster.

Emetics are not indicated by the disease, and should only be used where the abscess cannot be opened any other way. Laxatives are more advisable, especially where there are marked symptoms of cerebral hyperæmia.

Purgatives, mustard-plasters, foot-baths, as well as some remedies called specifics (tincture of pimpinella, borax, guaiac), have no effect on the disease.

In *chronic* parenchymatous angina, internal remedies are of no avail. As long as the swelling of the tonsils depends on their infiltration, we may paint solutions of alum, nitrate of silver, or dilute tincture of iodine on them, and apply cold compresses to the throat. Any remaining hypertrophy of the tonsils can only be removed by operation.

## CHAPTER V.

### SYPHILITIC AFFECTIONS OF THE PHARYNX.

ETIOLOGY.—The disturbances of nutrition in the pharyngeal tissue, caused by syphilis, occasionally consist only in hyperæmia, swelling, succulence, and perverted secretion of the mucous membrane, that is, in the characteristic symptoms of catarrh. In other cases, as a result of infection with syphilitic poison, we find the mucous papules, described when speaking of syphilitic affections of the mouth, which afterward become superficial ulcers or condylomata. Lastly we have, in the fauces and pharynx, gummy tumors, nodular tumors, and, by the breaking down of these, deep and often extensive loss of substance.

As syphilitic catarrh of the pharynx and syphilitic mucous papules come soon after the infection, they are classed among the secondary symptoms, while the gummy tumors, which do not appear till late, are classed among the tertiary symptoms.

ANATOMICAL APPEARANCES.—Syphilitic catarrh of the pharynx particularly affects the soft palate and tonsils. The generally sharp boundary of the redness, at the line where the soft palate becomes the hard, is as little characteristic of this disease as is a bluish-red (copper) color of the mucous membrane; we find both of these appearances in non-syphilitic cases of catarrhal angina.

Syphilitic mucous papules also come chiefly on the arches of the palate and the tonsils, which are sometimes extensively covered with them. In such cases, if the epithelial covering be milky, on superficial observation it looks as if the mucous membrane were covered with a croup membrane, and, if the white coating be present only in the space between the half arches of the palate, it seems as if there were an ulcer covered with a fatty base. The ulcers caused by the breaking down of syphilitic papules present losses of substance, reddened or covered with gray detritus, and bleeding easily, which gradually spread, by the breaking down of more recent papules that come around the edges, but show no tendency to become deeper. Condylomata form, small pedunculated excrescences, particularly on the uvula.

Gummy tumors occur in all parts of the pharynx. If they develop on the tonsils, these at first appear decidedly swollen, with smooth red surfaces. The breaking down of the nodules causes deep ulcers, of the size of a pea or a bean, with fatty floors. Not unfrequently gummy tumors form on the posterior wall of the velum, and then sometimes cause perforation before they are recognized. Gummy nodules, and the ulcers caused by their breaking down, occur most frequently on the uvula and the parts of the soft palate bordering it. At first the uvula looks as if gnawed, later it only hangs by a small pedicle, finally it and a large part of the soft palate may be destroyed. Under proper treatment, gummy tumors may be resolved. In such cases there is proliferation of connective tissue at the former seat of the nodule; this subsequently shrinks, and there is a cicatricial contraction. If extensive ulcers heal, there remain radiated, firm, white cicatrices—occasionally also adhesions of the soft palate to neighboring parts, constrictions and distortions of the pharynx, or closure of the Eustachian tube.

Symptoms and Course.—Syphilitic catarrh of the pharynx cannot at first be distinguished from other pharyngeal catarrhs; diagnosis is only possible later in the disease. If a patient has had difficulty of swallowing for weeks, if this difficulty has come on gradually, not suddenly, and if it obstinately resists all treatment, we may strongly suspect that the existing catarrh is of syphilitic nature. If these difficulties are found in a person who had a chancre a few weeks previously, and if they improve rapidly under the use of mercurials, the diagnosis may be considered as certain.

Syphilitic mucous papules often develop without pain or other inconvenience. Sometimes we find them accidentally, when examining the throat of a patient who has other symptoms of syphilis. If they have changed to ulcers, they cause pain in swallowing. The objective symptoms are given above.

Gummy tumors do not cause pain or difficulty of swallowing till they have softened and ulcerated. When patients that we suspect of syphilis complain of difficulty in swallowing, we should never neglect to examine the posterior surface of the velum with the finger or the rhinoscope, when inspecting the throat. Occasionally our attention is called to ulcerated nodules at the above locality, by a circumscribed dark-red spot on the anterior surface of the velum. The acts of swallowing and speaking are impaired, as before described, by perforation of the velum; this impairment is the greater the farther forward the perforation has occurred. In eating and drinking, solids and fluids return into the nose; and as soon as the patient speaks, we hear the nasal twang to his voice. For the objective symptoms, we may refer to the last paragraph.

TREATMENT.—Syphilitic affections of the throat must be treated according to the rules to be hereafter laid down when speaking of syphilis. In recent cases, the favorable action of mercurials is very striking. When there is danger in delay, I often employ *Weinhold's* treatment (which is of late very unpopular), with the modification that, for several evenings in succession, I give ten to twenty grains of calomel, until the ulceration is arrested, which is usually by the third or fourth day.

## CHAPTER VI.

### RETROPHARYNGEAL ABSCESS.

ETIOLOGY.—Inflammations terminating in suppuration are occasionally seen, especially among children, in the connective tissue between the spinal column and the pharynx. This affection is usually caused by caries of the spine, or a "scrofulous" inflammation and suppuration of the lymphatic glands at the back of the pharynx; at other times it develops with secondary inflammation of other organs, late in typhus, measles, the septicæmiæ, and other infectious diseases; lastly, it appears to occur occasionally as an idiopathic inflammation.

ANATOMICAL APPEARANCES.—The posterior wall of the pharynx is often pressed forward by the collection of pus, and the pharynx contracted or entirely closed; the pus may subsequently perforate the wall of the pharynx, or even sink into the breast, and there perforate the œsophagus, trachea, or pleura.

SYMPTOMS AND COURSE.—When disease of the cervical vertebræ accompanies retropharyngeal abscess, the affection is preceded for a time by peculiar stiffness of the neck and other symptoms of the vertebral disease; in this case we cannot easily make a mistake, for, as soon as there is difficulty of swallowing, the inside of the throat will be carefully examined. It is otherwise, especially in small children, when the affection begins without these preliminary symptoms. The restlessness of the child, its refusal to take the breast, its anxiety when compelled to drink, and the attacks of coughing and choking which interrupt the drinking, are occasionally referred to some primary affection of the larynx, as croup, laryngismus, etc. This is particularly liable to be the case when, besides the above symptoms, there is continued dyspnœa, the child is hoarse or voiceless, and the cough has a croupy sound. With the above symptoms it would be unpardonable not to examine the pharynx carefully; this examination quickly certifies the diagnosis: the finger usually encounters, close behind the soft palate, a tense, elastic tumor, which usually fluctuates distinctly and cannot be readily mistaken. Sometimes the abscess breaks spontaneously into the pharynx, its contents being swallowed or vomited up, and there is immediate relief of the symptoms. More frequently, if aid be not given at the proper time, the patient dies. There may be complete closure of the glottis by the swelling or the occurrence of œdema glottidis, or the opening of the abscess during sleep, and the entrance of its contents into the larynx, may choke the patient. In other cases the abscess sinks into the breast and causes pleuritis, pneumonia, pericarditis, etc.

TREATMENT.—The abscess is to be opened as early as possible. My old preceptor, *Peter Kruckenberg*, of Halle, said, in his humorous way, which always had a substratum of earnest, that every physician should allow one of his finger-nails to grow long, and sharpen it like a lancet, so that it would be always ready to open immediately any retropharyngeal abscess that he might run against. Probably none of his pupils ever followed this advice of "old Peter," but doubtless some of them have to thank him for the symptoms of retropharyngeal abscess always remaining fresh in their minds, and that no cases of it have escaped them.

## CHAPTER VII.

### ANGINA LUDOVICI.

THE floor of the mouth and the intermuscular and subcutaneous connective tissue of the submaxillary region are occasionally the seat of a phlegmonous inflammation, which may readily lead to diffuse gangrene and sloughing, but in other cases ends in formation of abscess or not unfrequently in resolution. This disease (which is often improperly called "gangrenous" inflammation of the neck, and which we name Angina Ludovici, after the deceased *Ludwig*, of Stuttgard, who first fully described it) is said by reliable observers to occur as a primary and idiopathic disease, and sometimes to be epidemic. In the few cases that I have observed, the inflammation of the connective tissue undoubtedly proceeded from periostitis of the lower jaw. Lastly, there is a form of the disease which comes with symptomatic or metastatic parotitis occurring in typhus and other infectious diseases; this probably starts from the submaxillary glands.

The disease begins with a more or less painful, very hard swelling in the vicinity of one or other submaxillary gland. We may feel this swelling from the mouth, as well as from the outside; the skin covering it is of normal color. The swelling soon extends over the entire submaxillary region, and upward toward the parotid; sometimes also downward, toward the larynx and trachea. The floor of the mouth is pressed far upward. Chewing and speaking become very difficult; the movements of the tongue are almost arrested, and the patient cannot open the mouth, because the muscles by which this is done are partly embedded in the infiltrated cellular tissue, and partly participate in the inflammation. The affection is usually accompanied by moderate fever and slight general disturbance; at other times the fever is high, and there is great constitutional sympathy.

Even in favorable cases, when resolution has occurred, the hardness disappears very slowly. When an abscess forms, the skin becomes red at some points, and fluctuation occurs; finally the pus breaks through the thinned covering. Quite as often the abscess opens into the mouth. When it terminates in gangrene, and there is perforation, instead of healthy pus, we have a fetid, discolored fluid, containing shreds of tissue. Death may result, at the height of the disease, from œdema glottidis and suffocation; at the termination, in gangrene from septicæmia; in the metastatic forms death usually results from the original disease.

At the commencement of the affection we attempt to secure resolution by the application of a large number of leeches near the tumor. Later we should continuously apply warm cataplasms. As soon as there is fluctuation we evacuate the matter through a large incision. Where there is danger of suffocation, we should scarify freely, and, if this does not answer, we should proceed to tracheotomy. If a hard, indolent swelling of the maxillary region remain for a long while, I find that repeated blisters do more good than rubbing in iodine or mercurial salves, or painting on tincture of iodine or *Lugol's* solution.

# SECTION III.

## *AFFECTIONS OF THE ŒSOPHAGUS.*

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## CHAPTER I.

### INFLAMMATION OF THE ŒSOPHAGUS—ŒSOPHAGITIS—DYSPHAGIA INFLAMMATORIA.

**Etiology.**—Catarrhal, croupous (diphtheritic), and pustular inflammations may affect the mucous membrane of the œsophagus, which may also be the seat of ulcers, or even mortify from the action of strong chemical agents; lastly, there are inflammations and suppurations of the submucous tissue.

*Catarrhal* inflammation is most frequently caused by the action of local irritants, such as acrid or too hot food, awkwardly-introduced œsophageal sounds; in other cases, the catarrh extends from the stomach or pharynx to the œsophagus; in still others, it may depend on venous congestion, which, in diseases of the heart and lungs, often exists throughout the whole intestinal canal.

*Croupous* inflammation of the œsophagus is rarely seen, and, when it does occur, it is almost always in company with similar inflammations of the larynx and pharynx, or in protracted typhus, cholera, and the acute exanthemata.

*Pustular* inflammation comes in some rare cases of variola, or after the use of tartar emetic.

*Ulcers* of the œsophagus are mostly caused by pointed bodies, which penetrate the mucous membrane, or by angular bodies that have become lodged at some spot in the œsophagus; more rarely, it comes from corrosion of the mucous membrane, or in the course of chronic catarrh. The same causes may excite inflammations and suppurations of the submucous tissue.

Lastly, the mucous membrane is sometimes *burned* by corrosive substances, particularly by concentrated acids.

**Anatomical Appearances.**—Acute catarrhal inflammation of the œsophagus is rarely found on *post-mortem* examination; when it is seen, the mucous membrane appears very red, swollen, readily torn, and is covered with a mucous secretion. In chronic catarrh, the mucous membrane, particularly that of the lower third of the œsophagus, appears thickened, dirty brown, or slate-gray, and is covered with tough mucus. Chronic catarrh may cause *dilatation* of the œsophagus by relaxation of its muscles, or *stricture*, by partial hypertrophy of the muscles and submucous tissue (see Chapter III.).

In *croupous* inflammation of the œsophagus we find the mucous membrane dark red, and covered with thick layers of exudation, in spots, or spread out widely.

In *pustular* inflammation slight elevations form, fill with pus, burst, and leave a superficial loss of substance; when caused by tartar emetic, the disease is limited to the lower third of the œsophagus.

*Ulcers* of the œsophagus are mostly superficial excoriations, but they may also destroy the entire thickness of the mucous membrane, and attack the muscles and surrounding connective tissue. When chronic, inflammation of the submucous tissue may lead to thickening of the walls of the œsophagus and stricture; when acute, it may terminate in abscess.

In inflammation of the œsophagus from corrosive substances, the parts affected are changed to a discolored, brown, or black slough, in whose vicinity injection and extensive serous exudation are quickly developed. The sloughs become detached, the loss of substance may be filled up; if the destruction was extensive, stricture of the œsophagus always remains as a result of the contraction of the cicatricial tissue.

Symptoms and Course.—In swallowing a hot mouthful, we may notice how little sensibility the œsophagus has, particularly at the lower portion. Hence we only have pain in very severe inflammations of the œsophagus, when caused by burns, injuries from pointed or angular bodies, but particularly after corrosion from caustic substances. This pain is felt deep in the breast, and at the back, between the shoulder-blades. In these cases we also find difficulty of swallowing; for, as soon as the muscles of the œsophagus are inflamed or infiltrated with serum, they cannot pass the morsel downward. This condition, which was formerly described as dysphagia inflammatoria, is always accompanied by oppression and great anxiety. The higher up the morsel is arrested, the more distinctly the patient feels it. If he makes new attempts to swallow, the contractions of the œsophagus may drive upward its contents, which cannot pass downward, so that there will be a regurgitation of the partly-swallowed substance, bloody mucus, and masses of exudation (see Chapter II.). These symptoms are always accompanied by excessive thirst, and, where the inflammation is extensive, there may also be fever. When the disease runs a favorable course, the symptoms disappear gradually; after the perforation of a submucous abscess, they may pass away suddenly; in other cases stricture remains; occasionally, even death is caused by perforation or rupture of the œsophagus (see Chapter V.).

During life, the slighter cases of acute and chronic catarrh do not have any recognizable symptoms. The same is true of pustular inflammation. The croupous form also is usually overlooked, unless pseudomembranes are vomited up; if it accompanies croup of the larynx and fauces, the dyspnœa and other symptoms of these affections throw into the background the pain and difficulty of swallowing; when it comes as a secondary croup in typhus and similar diseases, the patients usually lie in a perfectly apathetic state, so that they utter no complaints.

Chronic ulcers occasionally cause pain at some circumscribed spot, and permanently interfere with swallowing; they can only be distinguished from strictures by introducing an œsophageal bougie, which, in case of ulcers, finds no obstruction, and often brings up mucous, bloody masses. As the ulcers cicatrize, the symptoms of stricture may occur.

TREATMENT.—The question of treatment can only arise in the more severe forms of œsophagitis, as the slighter cases are not recognized. Foreign bodies exciting the inflammation are to be removed according to the laws of surgery. In corrosion by mineral acids and caustic alkalies, antidotes can only be used in very recent cases. For the rest, in acute catarrh, we may limit ourselves to giving the patient ice-water to swallow, or let him take pieces of ice in the mouth. General and local bleeding are only injurious; the employment of medicines is difficult, and promises little benefit. If the patient can swallow, he should take only fluids. If swallowing be totally impossible, the patient may be nourished through the stomach-tube, or by enemata. In chronic ulcers of the œsophagus, the numerous remedies recommended remain without effect, and careful nourishment of the patient is the chief object of treatment.

## CHAPTER II.

### STRICTURES OF THE ŒSOPHAGUS.

ETIOLOGY.—Contractions of the œsophagus may be due—1, to compression; 2, to the protrusion of new growths into its canal; 3, to structural changes of its walls. The latter form are strictures in the exact sense of the word; they result from the inflammations described in the last chapter.

ANATOMICAL APPEARANCES.—Compression of the œsophagus may arise in various ways. Among the most frequent causes we may mention: swelling of the thyroid bodies or of the lymphatic glands of the neck or mediastinum; dislocation of the hyoid bone; exostoses of the vertebræ; abscesses or tumors between the trachea and œsophagus; carcinoma of the lungs or pleura; aneurism. Not unfrequently the diverticuli, to be described in the next chapter, compress the section of the œsophagus immediately below them. In some cases where, during life, there were signs of compression of the œsophagus, on *post-mortem* examination, the right subclavian artery has been found morbidly dilated, arising from behind the left subclavian, and running to the right between the œsophagus and trachea, or œsophagus and vertebræ. The difficulty of swallowing thus caused has been named dysphagia lusoria.

In Chapter IV. we shall speak of the new formations on the inner wall of the œsophagus, which are the most frequent causes of its contraction.

Strictures of the œsophagus, in the exact sense of the word, depend—1, on cicatricial contractions of the membrane which have occurred after considerable losses of substance; they remain most frequently after corrosion or extensive ulceration; 2, on hypertrophy of the muscular and intermuscular connective tissue, induced by chronic catarrh of the œsophagus. On a longitudinal section through the wall of the œsophagus, which, in such cases, is frequently much thickened, there is often a peculiar fan-like appearance, as the hypertrophied muscular filaments are grayish red, while the hypertrophied connective tissue between them presents white fibrous bands, and the mucous membrane is thickened and irregular. Lastly, strictures may be due to hypertrophy and subsequent cicatricial shrinkage of the submucous tissue.

Sometimes the contraction is almost unnoticeable, at others so decided that the œsophagus is completely closed. The most frequent seat of stricture is the lower third, but it may occur in any part. Above the stricture, the walls are almost always hypertrophied, and the canal dilated; below it, the walls are often thinned, and the canal collapsed.

SYMPTOMS AND COURSE.—As strictures of the œsophagus from any cause develop gradually, the disease is at first apparently without danger, and does not cause much inconvenience. For a long time the sole symptom is a slight impediment in swallowing large morsels, which is overcome when the patient drinks or makes new efforts to swallow. Although the patients become more careful, and chew all their food very fine, they gradually find it more and more difficult to swallow. Even when the stricture is near the cardiac orifice of the stomach, they almost always indicate the region beneath the manubrium sterni as the place where the food sticks; finally, they cannot even swallow liquids.

The greater the obstacle, the less the patient succeeds in overcoming it by drinking, or by renewed attempts to swallow; and the more frequently the food regurgitates. An antiperistaltic movement, in which the contraction of a lower segment of the œsophagus is followed by the contraction of the segment just above it, has not been physiologically observed, it is true; on the contrary, the contractions which are voluntarily begun in the pharynx always go from above downward; but these facts do not exclude the possibility of a morsel of food, which cannot pass downward, being pressed upward by contractions which have proceeded peristaltically from above down to the point of stricture, or of a regurgitation in the same way, into the mouth, of the contents of the œsophagus, which has been filled up to a certain point. Occasionally there is no abdominal pressure in this form of vomiting; in other cases there is spasmodic contraction of the muscles of the abdomen without any influence on the evacuation of the œsophagus. When the contraction has increased still further, after every attempt to eat or drink; often after a few mouthfuls, occasionally not till a good deal has been swallowed (Chapter III.), there is a feeling of pressure deep in the breast, accompanied with great unpleasantness and anxiety, which increases until, with intentional or instinctive attempts to swallow, the food is slowly evacuated from the mouth, little changed, but largely mixed with mucus. The introduction of a bougie affords the best diagnostic sign, as it shows not only the existence of the stricture, but also its grade, locality, and even its form.

Besides the appearances described, and the other symptoms that a carcinoma or other tumor causes, the impaired nutrition induces gradual emaciation, and the belly sinks in; there may be no passage from the bowels for weeks, the patient starves, and, as *Boerhaave* aptly says, "tandem post Tantali pœnas diu toleratas lento marasmo contabescunt."

TREATMENT.—The treatment of stricture of the œsophagus belongs to surgery. By skill, patience, and persistence, surprising results are sometimes attained. In the surgical clinic at Griefswald there was a patient who, without perceptible cause, had a stricture of the œsophagus; at first only a common elastic catheter could be passed through it, but after four weeks it was so dilated that not only could the largest œsophageal sounds be passed, but ordinary morsels of food could be swallowed with ease.

## CHAPTER III.

### DILATATION OF THE ŒSOPHAGUS.

Etiology.—The dilatation of the œsophagus is sometimes *total*, affecting the entire organ, sometimes *partial*, limited to a short section. In partial dilatation sometimes only one wall is affected, then enlargements form which often develop to large sacs, communicating with the œsophagus; they are called diverticuli; their walls are sometimes formed of the mucous membrane, which protrudes hernia-like between the muscular filaments, and of the external connective tissue layer.

Besides the diverticuli, dilatations of the œsophagus are most frequently found:—1. Above a constricted portion; in stricture of the cardiac orifice there is total, when the stricture is higher up there is partial dilatation. 2. In other cases the total dilatation appears to depend on a chronic catarrh and on the muscular paralysis induced by it. 3. In many cases the causes are unknown. *Rokitansky's* hypothesis, that concussions of the body, and *Oppolzer's*, that the treatment of gout with large quantities of warm water, may cause enormous dilatation of the whole œsophagus, appear to me very problematical.

The diverticuli are formed—1. By foreign bodies which have stuck in the walls of the œsophagus, and are constantly driven farther in by the food which passes down. 2. They are sometimes formed by the shrinkage of bronchial glands, which have become adherent to the mucous membrane, while they were swollen, and which on contracting draw the mucous membrane after them. 3. In other cases we can discover no cause.

Anatomical Appearances.—In total dilatation of the œsophagus, the entire canal has been found dilated to the size of a man's arm; the walls are usually hypertrophied, more rarely thinned.

In partial dilatation, the portion immediately above the constriction is usually largest. The dilatation gradually decreases as we go upward, so that an elongated sac is formed, at whose fundus there is a second, narrow exit.

Diverticuli usually form near the bifurcation of the trachea, or at the point where the pharynx becomes the œsophagus; they are at first roundish, but later they form cylindrical or conical appendages to the œsophagus, lying between it and the spine. Such diverticuli sometimes only communicate with the œsophagus by a narrow fissure; in other cases, they seem to be prolongations of the œsophagus itself, with a blind end, which the food enters, while alongside of it the lower part of the œsophagus lies empty, constricted, collapsed.

Symptoms and Course.—Total dilatation exists without the presence of any symptoms by which the affection may be recognized. The partial dilatation that forms above a contracted part modifies the symptoms, so that the food remains in the œsophagus for a longer time and in greater quantities before regurgitating. When the food is finally vomited, it is softened, mixed with mucus, sometimes decomposed, but it is undigested and almost always of alkaline reaction. This circumstance may be useful in deciding whether the food comes from the stomach or œsophagus.

When the diverticuli are so large that food goes into them instead of into the stomach, they excite the same symptoms as stricture with partial dilatation. The food that has been swallowed sometimes regurgitates hours afterward, and may then be much decomposed, so that there will be a very bad smell from the mouth of the patient. Occasionally introducing the bougie renders the diagnosis certain, since we may at one time meet an insurmountable obstacle to its passage, while at another it may readily pass the diverticulum, and enter the stomach. If the diverticulum be at the commencement of the œsophagus, a soft tumor may be found in the neck behind the larynx, which increases in size after eating and drinking, and diminishes when the food and drink have been evacuated; if it be farther down, by pressure on the trachea and great vessels, it may cause dyspnœa, and disturbance of the circulation. In these cases, also, the patient may finally die of starvation.

TREATMENT is of no use in dilatation of the œsophagus. If we can pass a stomach tube through the diverticulum into the stomach, we may attempt to feed the patient in this way for a while, with a very slight hope that, if the food no longer enter the diverticulum, it may decrease in size.

## CHAPTER IV.

### MORBID GROWTHS IN THE ŒSOPHAGUS.

ETIOLOGY.—Fibroid tumors are rarely, and tubercles almost never, seen in the œsophagus; but carcinomata occur quite often. They are usually primary, more rarely carcinomatous growths spread from the mediastinum to the œsophagus.

The cause of cancerous degeneration of the œsophagus is just as unknown as that of cancer elsewhere. It has been claimed that brandy-drinkers are particularly liable to the disease.

ANATOMICAL APPEARANCES.—The fibroid tumors form movable, bluish-white concretions of the size of a lentil or bean, in the submucous tissue, or they appear as pedunculated polypi, often lobulated at the free end, which usually originate from the cricoid cartilage (*Rokitansky*).

Of the carcinomatous growths, scirrhus and medullary cancer, and very rarely epithelioma, occur in the œsophagus. They generally affect the upper or lower third, more rarely the middle third; the whole circumference is usually comprised in the degeneration forming a cancerous stricture. The degeneration always begins in the submucous tissue, but soon attacks the mucous membrane. If the cancer softens and disintegrates, uneven ulcers form, surrounded by a medullary infiltrated wall, and covered with sanies, and bleeding fungous growths, or black ragged masses. From the external connective tissue membrane of the œsophagus, the cancer may extend to the neighboring structures, and, when breaking down, cause perforation of the trachea, bronchi, or even of the aorta and pulmonary arteries.

SYMPTOMS AND COURSE.—The small movable fibroids of the œsophagus cause no symptoms; pedunculated fibrous polypi cause the symptoms of stricture of the œsophagus, and may induce hæmorrhage; the œsophageal sound may be passed around them, and, when they are high enough up, they may be reached with the finger.

Cancer of the œsophagus is not easily mistaken. If, in a person of advanced age, particularly in one who has been in the habit of drinking strong liquor, difficulty of swallowing gradually occurs, without any other known cause, and increases slowly till it produces the very painful symptoms described in the second chapter, we may very strongly suspect carcinoma, for we know that this is by far the most frequent cause of stricture of the œsophagus, and that all other forms are proportionately very rare. The presumption that the disease is cancerous increases in probability when there are lancinating pains at various places, particularly between the shoulder-blades, when the patient emaciates rapidly, and the dirty-yellow, cachectic appearance of the face, common to cancer-patients, occurs. The diagnosis becomes absolutely certain when we find fragments of cancer in the mucous, sanious, or bloody masses, that are vomited or brought up with the œsophageal sound. Subsequently, when the cancer sloughs, the symptoms of stricture subside; nevertheless, the emaciation continues, the feet swell, coagula often form in the femoral veins, and, finally, the patient dies from exhaustion, or from perforation of one of the above-named organs.

TREATMENT.—Dilatation of the cancerous stricture by bougies is dangerous, and should never be tried when the diagnosis is certain. In the earlier stages it may hasten the sloughing of the cancer, and later it may cause perforation of the œsophagus. The treatment must be symptomatic. If there is great pain, we may give opium; when there is inability to swallow, we may make the almost hopeless attempt to nourish the patient by enemata.

## CHAPTER V.

### PERFORATION AND RUPTURE OF THE ŒSOPHAGUS.

PERFORATION of the œsophagus may take place from within outward, or the reverse. The first form most frequently results from the breaking down of cancer, more rarely from ulcers caused by splinters of bone, or from deep sloughs, excited by corrosion with caustic substances. So-called perforating ulcers, such as are found in the stomach and duodenum, are never seen in the œsophagus. The œsophagus may be perforated from without inward by aneurisms of the aorta, by the breaking down of tuberculous bronchial glands, especially of those located at the bifurcation of the trachea, by abscesses on the anterior surface of the spine, by caries of the vertebræ, even by tuberculous cavities in the lungs, etc.

Rupture of the œsophagus without precedent disease has only been observed in a very few cases (*Boerhaave*, *Oppolzer*). It more frequently happens that the wall of the œsophagus, which has been almost destroyed by carcinoma, corrosion, or ulcers, and nearly perforated, is suddenly ruptured by severe retching and vomiting.

If the wall of the œsophagus is opened in any way, its contents pass into the surrounding connective tissue, or communication is opened with the trachea, pleural or pericardial sacs, or with the great vessels.

Before perforation or rupture of the œsophagus occurs, the advancing destruction may cause adhesive inflammation of the adjacent organs, the symptoms of which precede the perforation. I have seen double pleurisy and pericarditis gradually develop in a man with carcinoma of the œsophagus; on *post-mortem* examination I found the parts of the pleura and pericardium lying next the cancer discolored and mortified, but no escape of the contents of the œsophagus into those cavities. Sudden, severe pain, deep in the breast, usually indicates the moment of perforation; besides this there are chill, paleness, and coolness of the extremities, fainting, and sometimes, depending on the seat of the perforation, attacks of suffocation, or symptoms of severe pleurisy, or profuse vomiting of blood. Death sometimes occurs immediately. There can be no treatment.

## CHAPTER VI.

### NERVOUS AFFECTIONS OF THE ŒSOPHAGUS.

Globus hystericus, or the feeling of a ball rising to a certain point in the œsophagus and remaining there, has been called *hyperæsthesia*, i. e., increased excitability of the sensory nerves, of the œsophagus. We have already mentioned globus hystericus when speaking of the nervous affections of the larynx. Some cases that are described as spasm of the œsophagus should also be reckoned among the hyperæsthesiæ; such as those where the patient feels as if the œsophagus were ligated, and thinks he cannot swallow. This state not unfrequently occurs in persons that have been bitten by dogs. *Andral* relates a case where *Boyer* was obliged to stay with a patient at meal-times for a whole month, because she thought she would suffocate as soon as she attempted to swallow.

There can hardly be *anæsthesia*, that is, diminished or lost excitability of the sensory nerves of the œsophagus, because the normal sensitiveness is so very slight.

*Hyperkinesis*, increased excitability of the motor nerves, œsophagismus, or dysphagia spastica, occurs more frequently, although, doubtless, many cases, classed under this head, have been misinterpreted. Spasm of the œsophagus is most frequently of reflex origin; it is often excited by irritation of the uterus, hence is most frequently met in hysterical women; occasionally it is of central origin and forms one symptom of disease of the brain or upper part of the spinal marrow; it may also be induced by poisoning with narcotic substances or alcohol. Like most neuroses, spasm of the œsophagus runs its course with paroxysms and free intervals. The attacks most frequently occur during eating; the patient suddenly becomes unable to swallow, and feels as if there were a foreign body in the œsophagus. If the spasm be at the upper end of the organ, the food returns as soon as introduced; if at the lower end, it does not regurgitate for a short time. There are, usually, at the same time, attacks of oppression and suffocation, and sometimes spasmodic contractions of the muscles of the neck. After lasting for a while, the attack usually passes off; in other cases, a slight amount of spasm remains for weeks or months as a permanent affection, called "spastic stricture." During the interval, if we examine with the bougie, we find no obstacle; if we examine during the attack, the stricture occasionally disappears during the probing. Besides a proper treatment of the original disease, it is advisable to use narcotics, particularly belladonna, or the so-called antispasmodics, such as valerian, asafœtida, musk, etc. If the patient cannot swallow, these remedies should be used by enema. Repeated careful introduction of the œsophageal bougie promises the best results.

*Akinesis*, diminished excitability of the motor nerves of the œsophagus, is not unfrequently seen along with the signs of general paralysis shortly before death. In other cases, the paralysis is of central origin, and accompanies diseases of the brain, or of the cervical portion of the spinal marrow. In complete paralysis of the œsophagus, swallowing is impossible; often, when the bystanders wish to refresh the dying patient, they are horrified that he cannot swallow, and because the food or drink they offer returns out of the mouth, or passes into the larynx, and excites attacks of suffocation. When the paralysis is incomplete, food does not return, but large morsels and firm substances are most easily swallowed. Swallowing is facilitated by the upright position and by drinking. In this dysphagia the patient does not usually complain of pain, and the probe meets no obstacle. Treatment is almost always hopeless, on account of the severity of the original disease. The repeated use of the probe, the employment of strychnia and electricity, have been recommended, and it has been claimed that they have sometimes proved serviceable.

# SECTION IV.

## *DISEASES OF THE STOMACH.*

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### CHAPTER I.

### ACUTE CATARRHAL INFLAMMATION OF THE MUCOUS MEMBRANE OF THE STOMACH—ACUTE GASTRIC CATARRH.

ETIOLOGY.—During normal digestion changes occur in the gastric mucous membrane, which, if found in other mucous membranes, would be called catarrh. The secretion of the gastric juice is always accompanied by considerable hyperæmia of the mucous membrane, which is regularly followed by an abundant flow of mucus, and a considerable detachment of epithelium. This physiological process, like the analogous pathological one, is accompanied by a slight general disturbance, the so-called digestive fever. Hence, the definition, that we have given for catarrh of mucous membranes generally, does not answer for gastric catarrh; what in them is pathological is here normal, and we can only speak of gastric catarrh when the physiological process increases beyond normal bounds. It will be readily understood that, as the act of digestion is repeated several times during the day, and our food is complicated and sometimes of improper character, the process may readily become abnormal; hence, as may easily be conceived, acute gastric catarrh is one of the most frequent of diseases. On the other hand, it is just as evident that a morbid augmentation of normal processes may subside more readily and quickly than other more material deviations from the normal state. Hence, under favorable circumstances, gastric catarrh usually lasts a shorter time than that of other mucous membranes.

The *predisposition* to this affection varies with the individual; in some persons it is induced by exciting causes, which would have no effect on others. In many cases increased predisposition to gastric catarrh depends on too scanty a secretion of gastric juice, as this favors abnormal decomposition in the stomach, which is the most frequent cause of the disease. On this diminution of the gastric juice depends the great inclination to gastric catarrh observed:

1. In all fever patients. It is going too far to say that every fever is accompanied by catarrh of the stomach; neither the coated tongue nor the loss of appetite of fever patients justifies this view. But, as in every fever, in consequence of the increased temperature, the amount of water lost through the skin and lungs is excessively increased, it may be concluded *a priori* that less gastric juice will be secreted; this supposition is confirmed not only by the analogous condition of other secretions, but by actual observations (*Beaumont*). (It is possible that in fever the composition of the gastric juice is also changed; but this hypothesis is not necessary to explain the results of slight errors of diet on the part of the fever patients.) If the patients do not bear this in mind, and adapt their diet to the diminished secretion of the stomach, very distressing gastric catarrh will result. A large portion of the gastric complications in pneumonia and other inflammatory affections result from neglect of this simple dietetic rule.

2. The increased predisposition to acute gastric catarrh, which we see in debilitated and badly-nourished persons, appears also to depend on diminished quantity or inferior quality of gastric juice, which favors the decomposition of the ingesta. If the amount of blood be decreased, it is probable that the quantity of gastric juice as well as of the other secretions is diminished. As, in hydræmia, there is a diminution of the albuminates of the blood, which we must regard as the material of which pepsin, the organic constituent of the gastric juice, is formed, the supposition is warranted that a juice, deficient in pepsin, is formed in such cases. From the diminished action of the gastric juice, part of the ingesta remain undissolved and decomposed; hence many convalescents have gastric catarrh from eating what would not have harmed them at another time. In the same way puny children have this disease when they take the same amount of mother's milk, or the same quantity of cow's milk diluted to the same extent, as healthy children of the same age can take without harm.

3. Although we have many analogous facts in other organs, it is not easy to explain the increased predisposition to gastric catarrh in persons who are very careful about their stomach, and carefully protect it from irritation. Catarrh of the stomach is more readily induced by a slight excess in drinking, in persons unaccustomed to the use of liquor, than in those who take a moderate amount daily; and by a slight error of diet in children whose diet is usually carefully watched, than in those accustomed to complicated and indigestible food.

4. Lastly, we find an increased predisposition to gastric catarrh in persons who have suffered from it repeatedly.

1. Among its *exciting causes* is the use of very large quantities of food, even of that which is very easily digested. We have already pointed out that in these cases acute gastric catarrh is not induced so much by the overfilling of the stomach as by the action of the products of decomposition, formed when the gastric juice does not suffice for the substances to be digested. Hence, after overloading the stomach, the symptoms of acute catarrh do not occur immediately, but come on next day.

In grown-up and sensible people it does not often happen that they have simply eaten *too much;* this is far more frequently seen in children, especially among such as have their diet very much restricted, and hence are never satisfied, but seize every opportunity to overload the stomach. Children at the breast hardly have any feeling of satiety; when nourishment is plenty, they usually drink till the stomach is overfilled. If they vomit easily, the overloading is soon removed, and only so much nourishment remains as they can readily digest; if they do not vomit easily, the stomach remains overfilled, and they are affected with gastric catarrh, although they have taken the most suitable nourishment. Nurses know very well that children which vomit often and easily ("spei-kinder") sicken less readily and thrive better than others.

2. Gastric catarrh may be excited by moderate use of food difficult of digestion. In this case, also, it is not the food itself, but the products of its decomposition, when partly undigested, that cause the difficulty. The indigestibility of food often depends on its shape. Persons who eat with avidity, or who have no teeth, often introduce perfectly digestible food into their stomachs in a state which offers little surface to the gastric juice, which is consequently slowly absorbed and digestion is retarded. It is well known that the yolk of a hard-boiled egg is far more easily digested than the white; this is simply because the former is far more readily broken into fine morsels in the mouth than the latter is. The use of fat meat, or greasy sauces mixed with the meat, often causes gastric catarrh, not, as the laity suppose, because fat is indigestible, but because, when mixed with the meat, it hinders its imbibition and so diminishes its digestibility. It would lead us too far if we were to mention all the substances that are indigestible, and may cause gastric catarrh, even when used in moderate quantities.

3. Gastric catarrh is often caused by the use of substances that have begun to decompose before entering the stomach. It may be thus caused in adults by spoiled meat, or by new beer; but it most frequently occurs in children from the use of milk that has begun to sour. This is what renders the artificial nourishment of infants so difficult in hot weather, when milk begins to spoil very soon. If children

do not have their mouths regularly cleaned, or if a sugar-teat be given them to prevent their crying, the decomposition of good fresh cow's milk, or even of the mother's milk, may be commenced in the mouth itself. (It is well known how carefully milk-cans must be cleansed and purified of all decomposing substances in order to prevent the milk from spoiling.) If decomposition has once begun in the milk in the stomach, the best milk taken subsequently will act as a poison, as it also soon begins to decompose. We shall hereafter see that fermenting substances in the stomach, after death, may destroy and dissolve its walls. Even if such an action on the walls of the stomach be prevented during life by the circulation and the rapid change of tissue in them, it is nevertheless not improbable that the epithelium, where nutrition is less active, may be destroyed, even during life, by the fermenting substance; and that the deprivation of the mucous membrane of its protection may cause extensive transudations. It appears not to be the lactic acid, the product of the souring of the milk, but the process of fermentation itself, which excites the symptoms of cholera infantum, and after death causes the softening of the stomach. We come to this conclusion because milk, which has already curdled, and whose sugar has been transformed into lactic acid, may be eaten even in large quantities by older children and by adults, without deleterious influence; and because the so-called softening of the stomach may be more readily induced in that organ, when cut out of an animal, by filling it with fresh milk and exposing it to a moderate temperature, than by filling it with dilute acid.

4. Acute catarrh of the stomach may also be caused by irritation, from taking into it very hot or cold articles, some medicines, alcohol, or spices. Alcohol acts most injuriously when it is but slightly diluted. Spices and similar substances, in small quantities, excite the normal processes, and hence may improve digestion; in larger quantities, however, they increase these processes beyond the normal limits, and lead to gastric catarrh.

5. Acute gastric catarrh is excited by the introduction of substances that weaken the digestive power of the gastric juice, or retard the movements of the stomach. It is evident that, in either case, there may be abnormal decomposition of the contents of the stomach. Apart from the direct irritation of the gastric mucous membrane, the misuse of alcoholic stimulants acts injuriously in this way. In the matter vomited the day after a debauch, much to the astonishment of the patient, he often finds some of the food eaten the previous day, which is hardly changed. The narcotics, particularly opium, seem to cause the gastric catarrh, which is so often seen after large doses of them, by impairing the movements of the stomach and thus preventing the food from

being sufficiently mixed with gastric juice, and keeping it too long in the organ.

6. Catching cold also leads to gastric catarrh, though less frequently than to catarrh of the respiratory organs.

7. Lastly, at certain times, without known cause, from a "genius epidemicus gastricus," gastric catarrh occurs surprisingly often; and at such times other affections are complicated with it, without there having been any error of diet. In this class belong the feverish, gastric, and intestinal catarrhs and cholera morbus, which are occasionally epidemic.

When speaking of infectious diseases, we shall treat of those cases of gastric catarrh which, like other catarrhs, are symptomatic of an infection.

ANATOMICAL APPEARANCES.—We seldom have the opportunity of seeing the remains of acute gastric catarrh on *post-mortem* examination; where we do, the gastric mucous membrane is found reddened in spots by a fine injection, its tissue is relaxed, and its surface covered with a layer of tough mucus. But more frequently, especially among children who die with the symptoms of cholera infantum, the autopsy gives negative results, except as to appearances that will be described hereafter. This does not appear strange, when we remember that the capillary hyperæmias of other mucous membranes, which we have been able to observe directly during life, leave no trace after death; and that a relaxation and partial loss of epithelium, which we have regarded as the most probable cause of the extensive transudation in cholera infantum, may be very readily overlooked in the dead body, and can very rarely be observed with certainty. Hence, the observations that *Beaumont* made on his Canadian *St. Martin*, when he had catarrh of the stomach after overloading that organ with indigestible substances, or after the excessive use of liquor, are very important. At the commencement of the affection the gastric mucous membrane appeared intensely reddened, had aphthous (?) spots on it, and was covered with tough mucus, here and there mixed with traces of blood. Later, the mucous covering was thicker, and the secretion of gastric juice was suppressed. The fluid taken out through the fistula consisted mostly of mucus and muco-pus, which showed an *alkaline* reaction. In a few days the mucous secretion and the alkaline reaction of the contents of the stomach ceased; and, at the same time, the mucous membrane regained its normal appearance.

The gastromalacia, or softening of the walls of the stomach, found on autopsy of children, was often diagnosticated during life, so that it appeared as if the diagnosis were confirmed by the *post mortem*. An exhaustive description of the symptoms of gastromalacia has also been given (*Jaeger*), and we often meet with cases answering the description. Nevertheless, there can be no doubt (*Elsaesser*) that gastromalacia is always a *post-mortem* appearance. The description of softening of the stomach is precisely that of cholera infantum, and thus there is a simple explanation of the apparent confirmation of the diagnosis by the autopsy. For, if a child dies who has had vomiting and purging from abnormal fermentation in the stomach, and if there are still fermenting substances left there, the fermentation will not be arrested by the gradual cooling of the body. When the circulation ceases, the stomach can no longer resist the decomposition, which then extends to it also, just as the stomach, that has been cut out of an animal and filled with milk, softens if left only for a short time in a warm place. Hence physicians, who consider softening of the stomach as a *post-mortem* appearance, may also predict it with certainty, when a child that has died of cholera infantum had eaten milk, or any other easily-decomposed substance, shortly before death.

*Rokitansky*, who does not consider softening of the stomach as a *post-mortem* appearance in all cases, distinguishes two forms, the *gelatinous* and the *black*. According to his description, the former almost always begins at the fundus of the stomach, and gradually extends along the greater curvature; the mucous membrane is first softened, but the softening soon extends to the muscular coat, and finally to the peritonæum. The membranes altogether change to a grayish or yellowish-red translucent gelatin, which occasionally has some blackish-brown striæ through it; these are the blood-vessels that are also softened. When the softened inner layer is detached, the fundus consists of a thin, easily-torn peritonæum. The softened stomach tears on the slightest touch, and comes to pieces between the fingers, or else we find that rupture has already occurred, and the contents have escaped into the abdomen. The process is not always limited to the stomach, but may attack the neighboring organs, especially the diaphragm; this may even be perforated, and the contents of the stomach may pass into the left side of the thorax. In the *black* softening of the stomach, the walls are not changed to a translucent gelatin, but to a blackish brown or black pulp. This modification occurs if the capillaries of the stomach are overfilled when the softening begins. The blackish-brown striæ in the gelatinous softening represent the same changes of the large vessels and of the blood contained in them, which, in this case, affect the capillaries and their contents.

The theory that gastromalacia does not occur till after death, or, at least, till a short time before it, when the circulation and the change of tissue in the walls of the stomach have almost ceased, is supported—1, by the fact that the softening is almost always found in the fundus of the stomach, where the acid contents are collected together, and it only attacks the pyloric portion, when, from the position of the body on the right side, the contents have settled to that portion; 2, by the circumstance that it is also found in the bodies of children who showed no signs of gastric disturbance during life, but who had taken milk, sugar-water, or other easily-fermenting substances, during the last hours of life; 3, because, even in cases where the walls of the stomach are found torn, and its contents have entered the abdominal cavity, there have been no symptoms of peritonitis during life, nor have any remains of it been found on autopsy; finally, 4, another proof is the above-mentioned experiments, where artificial softening was induced in stomachs that had been removed from animals.

(The cases where softening of the stomach has been found, while that organ was empty, do not belong here. It has been attempted to explain this by citing the digestive power of the gastric juice, and asserting that there was a self-digestion of the stomach, and that the gastric juice secreted shortly before death had digested the stomach just as it would digest any other membranous tissue. It is, however, improbable that gastric juice would be secreted into an empty stomach, and it is possible that a decomposition of mucus (which also sets free lactic acid) would have the same effect on the walls of the stomach as fermenting ingesta do.)

SYMPTOMS AND COURSE.—We shall first speak of the symptoms of acute gastric catarrh when it is accompanied by moderate fever, and often constitutes only an ephemeral affection. This form, the most frequent result of errors of diet, is usually called status gastricus, gastricismus, gastrosis, "disordered stomach."

Even the physiological process of digestion is accompanied by a certain depression, sluggishness, and disinclination to bodily or mental exertion; and the hyperæmia and production of mucus, when increased to acute catarrh, are accompanied by a general *malaise* and sick feeling that seem out of proportion to the slight and evanescent disease. The patients feel dull, are fretful, complain alternately of heat and cold; have a hot head, cold extremities, but particularly a pressing, tormenting pain in the forehead, which extends toward the occiput; on stooping, they have flashes before their eyes, and feel as if their heads would burst. The affection of the mucous membrane of the stomach causes a feeling of pressure and fulness, even when that organ is empty; the "pit of the stomach" is sensitive to pressure; there is loss of appetite, but increased thirst; there is usually distaste for food, and qualmishness. Besides these, there are symptoms caused by abnormal decomposition of the contents of the stomach; gastric catarrh is often the result of abnormal decomposition of the ingesta, and, on the other hand, it is sometimes the cause. *Bidder* and *Schmidt* have shown that when the gastric juice is rendered alkaline by admixture of mucus, it loses its power of dissolving protein substances, which then undergo spontaneous decomposition, and give out a putrid odor. Daily experience in practice confirms this experiment. But those substances also, that are not digested by the gastric juice, undergo abnormal decomposition in gastric catarrh. The amylaceous substances, whose change had already begun in the mouth from the admixture of saliva, under normal circumstances, are not converted into sugar until they enter the stomach. But, in gastric catarrh, the mucus secreted acts as a ferment, and induces a change of a large portion of the sugar into lactic acid, and often also into butyric acid. If, during gastric catarrh, fermented substances, such as beer or wine, be taken, or if excessive use of these has induced the affection, acetic fermentation takes place; if fatty substances be swallowed, fatty acids appear to be developed from them. In all of these decompositions of the contents of the stomach, except the lactic acid fermentation, gases are set free. In the breaking up of albuminous substances, stinking, sulphuretted, hydrogen gases are freed; hydrogen and carbonic acid are formed in butyric-acid fermentation; in acetic fermentation, carbonic acid is freed. This explains why the epigastrium is slightly prominent in patients with acute catarrh, and why, from time to time, they belch up gases which sometimes smell disagreeably, at others are odorless, according to the quality of the food that has been taken. At the same time, sour or rancid substances often rise into the mouth.

Since gastric catarrh, as before mentioned, is usually complicated with oral catarrh, the tongue is generally coated, the taste stale and slimy, and there is a bad breath.

If the patients fast, and do not expose themselves to any new sources of injury until the stomach is able to fulfil its normal functions, the above symptoms usually disappear quickly. The abnormally-decomposed contents of the stomach pass through the pylorus into the intestine; there further decomposition seems to be arrested sometimes by the admixture of bile, but more frequently, although moderated, it still continues; the secretion of the irritated intestinal mucous membrane increases, the movements of the intestines are hastened, flatulence, rumbling, etc., with griping pains in the belly, occur, and are relieved by the passage of badly-smelling gas; finally, one or more pulpy stools occur, and the trouble ends. If the patient sleeps the following night, his general health is usually improved, or fully restored. We may also mention that, during the affection, the urine usually contains quantities of pigment and urates, and that herpetic vesicles not unfrequently come on the lips.

When the injuries that excite the acute gastric catarrh are more intense, or the patient more sensitive, there is greater nausea, which finally increases to retching and vomiting. By the latter the contents of the stomach are evacuated, more or less changed, with a very acid smell and taste, and usually mixed with quantities of mucus. The vomiting may be repeated at varying intervals; the longer it lasts, the more the matter vomited is mixed with bile, which gives it a bitter taste and green color. These severe forms of the status gastricus are almost always accompanied by great irritation of the intestinal mucous membrane. Then there is severe diarrhœa, by which green masses are passed, with or without pain. After the vomiting and purging, the patient is almost always relieved, and, although perhaps a little feeble, is usually well otherwise in a couple of days. In other cases, the vomiting and diarrhœa are very bad, and present the symptoms of cholera morbus.

By *cholera morbus* we mean that form of acute gastric catarrh which extends to the intestinal mucous membrane, and is characterized by profuse transudation of a fluid, containing little albumen, into the stomach and intestines. These watery transudations occur so frequently in the first stage of acute catarrhs of other mucous membrane, especially of the nasal, that we cannot hesitate to designate as a catarrh the gastric and intestinal affection, on which depend the symptoms of cholera morbus, and mostly, also, those of Asiatic cholera, which will be hereafter described, and which only leads to symptoms that other catarrhs do not have, on account of its extent.

The disease prevails most during the hot weather of summer, and then often attacks a number of persons simultaneously; it is more rarely excited, at other times, by errors of diet. The cholera attacks are rarely preceded by premonitory symptoms; on the contrary, the patient is usually attacked suddenly, often during the night, with a disagreeable feeling of pressure at the pit of the stomach, which is soon followed by nausea and vomiting. At first the food last eaten is vomited, little changed, but the vomiting is soon repeated, and quantities of a pale-yellow or greenish bitter fluid are thrown up. After this, or, in some cases, even previously, there are borbyrigmi, followed by pulpy stools, which soon become thin and liquid. In a short time enormous quantities of fluid are evacuated; the greater the amount, the less color it has, as the bile, even if of normal amount, no longer suffices to color all the transudation. The loss of water from the blood excites intense thirst, which is only temporarily quenched by large quantities of drink. The fluid taken into the stomach is rapidly evacuated, upward or downward, being voided every quarter of an hour, or oftener, as long as the diarrhœa and vomiting continue. The blood constantly becomes thicker; the secretions, particularly that of urine, are diminished, or cease entirely, for want of fluid to maintain them; the interstitial liquid is absorbed from all the tissues; hence the skin appears dry and shrivelled, the patient looks collapsed and disfigured, the nose is pointed, the eyes are sunken, because the connective tissue in the orbit has become dry, and has hence actually lost in volume. While there is rarely pain in the abdomen, there are very painful contractions of the muscles, especially of the calf of the leg. If these occur, and the evacuations of the patient consist only of colorless fluid, containing shreds of intestinal epithelium, so that they resemble rice, water, or oat-meal gruel, the cholera morbus will very much resemble Asiatic cholera; nevertheless, it rarely goes on to the complete disappearance of the heart-beat and the pulse, to the cyanotic hue, and reptile temperature of the skin, which is seen in the so-called asphyxiated stage of the Asiatic cholera. No matter how threatening the symptoms, how great the collapse and depression of the patient, how dispirited he and his attendants may appear, the physician must not feel discouraged if he is sure that epidemic cholera is not raging, for he must know that a previously healthy adult very seldom dies of cholera morbus. Usually, after a few hours, rarely not till the next day, the vomiting and purging subside; the skin becomes warm, and acquires its fulness again, the exhausted patient falls asleep, and only suffers from great depression. More rarely, the symptoms of gastric fever join on to the cholera morbus. In the rarest cases, and only in sickly and weak persons, or in children or old persons, do we see a fatal termination; then the bowels are paralyzed, the vomiting and purging cease, while the transudation continues; the pulse disappears, the movements of the heart become weaker, the intellect cloudy, and the patient dies of exhaustion.

The acute gastric catarrh of children, during the first years of life, presents certain peculiarities, which are due to the fact of such children being almost exclusively nourished with mother's or cow's milk. *Bednar* considers the fermentation of the ingesta as the sole cause of this disturbance of digestion, and denies either a primary or secondary participation of the walls of the stomach in the affection; he designates the milder forms of the affection as *dyspepsia;* according to the classic description of this author, the appearance of the child is little changed, at most it only looks a little pale and has a slight ring around the eyes. Almost always, shortly after nursing, there is vomiting, and the milk evacuated is no longer curdled. This sort of vomiting is an important symptom; even the nurses recognize it as such, and readily distinguish it from the healthy evacuations of an overfilled stomach. The curdling of the milk in the so-called "puking of children" does not show that the milk has become sour, but that the gastric juice has acted normally on it, and curdled the casein; when the vomited milk is not curdled, it shows that there is an abnormal secretion in the stomach, and this must excite the suspicion of gastric catarrh. Soon after the vomiting, or even at the same time, the passages from the bowels become abnormal, or there may be no vomiting, and the appearance of the passages may form the sole symptom of gastric catarrh. The evacuations consist of a very acid, green or greenish-yellow fluid, containing more or less firm lumps; they remind us of the changes that the milk undergoes after standing for some time out of the body, and show that the gastric juice has not even digested it enough to cause its sudden coagulation. The vomiting and purging, which are usually preceded by restlessness of the child, by crying and drawing the legs up toward the belly, occur more or less frequently; the evacuations often change their color and consistence. In many cases, the vomiting ceases after a few days, the undigested milk disappears from the evacuations, the children improve and pick up; but in other cases, from time to time, quantities of acid milk, partly unchanged, partly curdled and mixed with mucus, are vomited; the purging increases, the evacuations become thin, liquid, and very free; at first they are bright yellow or green, but at last almost white. Some yellow or greenish flocculi swim in the colorless fluid; these remain on the diaper, while the fluid partly filters through, partly leaves large, damp, discolored spots in it. Even now both the smell and reaction of the evacuations are acid. Occasionally the appearance of the dejections changes suddenly, without our being able to say why; they become dark brown or clayey, and softer masses of disagreeable smell are evacuated in large quantities. These severer forms of acute gastric and intestinal catarrh reduce the child rapidly; its face falls and is contracted with pain, it may even become wrinkled in a few days, the eyes are usually half opened and deep set, the lips as well as the hands and feet are often bluish, the rest of the body, especially the back, is mottled. The temperature is uneven, the trunk, especially the belly, is burning hot, while the face and limbs are cool. From the diminution of cerebral pressure the fontanelles become depressed, occasionally even the frontal and occipital bones sink slightly below the parietal bones, the movements of the children become sluggish, even nursing troubles them; they let go of the breast, but eagerly drink water when it is offered to them. The cries of pain which usually precede the evacuations gradually change to weak whimpering; in the interval the child lies half asleep. As the exhaustion increases, many die; occasionally, shortly before death, convulsions (hydrocephaloid) and other symptoms of anæmia of the brain appear. When the disease runs a favorable course, the evacuations gradually become fewer and more normal, the collapse disappears, the temperature becomes more even, the child improves and convalesces, but a great tendency to relapse remains.

If the symptoms above described appear very rapidly, and the evacuations come one right after the other, if decided collapse occurs in a few hours, with great depression of the bodily temperature, and signs of thickening of the blood, before emaciation has resulted, we call it *cholera infantum.* The thickening of the blood is shown by the unquenchable thirst; older children follow the glass of water with eager eyes, and when it is offered to them seize it with both hands and hold it tightly till it is emptied; it further betrays itself by the increasing cyanosis, and by a peculiar dyspnœa, in which the thorax and diaphragm make extensive movements, without there being any apparent obstruction to the breathing, except the difficulty of the thickened blood passing through the capillaries of the lungs. The patients may die in a few hours of *cholera infantum*, with the above symptoms; in other cases, the cholera proper passes off, and a milder form of the disease remains; and finally, in other cases, rapid and complete recovery take place from conditions which are apparently utterly hopeless.

DIAGNOSIS.—In Chapter X. of this section we shall speak of the distinction between gastric catarrhs, occurring as the *status gastricus*, and other disturbances of digestion.

During an epidemic of Asiatic cholera it is impossible to distinguish cases of *cholera morbus* from those caused by the cholera miasm, for the symptoms are not only similar, but are absolutely the same as those of the milder cases of Asiatic cholera. The chief difference is, that of those attacked with Asiatic cholera about half the patients die, while almost all recover from cholera morbus. The disease may much more readily be mistaken for poisoning; but cholera morbus is rarely accompanied by such severe pain as poisoning with acids and metallic salts induce, and they seldom cause such copious evacuations as characterize cholera morbus. If the disease lasts unusually long, or if its course shows any other peculiarity, we should carefully examine every circumstance that could indicate the presence of poison.

Acute gastric catarrh of children in the first years of life and the diarrhœa of children cannot easily be mistaken for other diseases.

PROGNOSIS.—The prognosis is evident from the description we have just given of the course. Previously healthy adults rarely die of this disease; but chronic catarrh may result from repeated attacks, weakly and decrepit persons may die of gastric fever, or still more readily of catarrhal fever (see this disease). In children, acute gastric catarrh, with its results, is a very dangerous disease, which may end fatally even under the most careful treatment.

TREATMENT.—To speak with only moderate exactness of acute gastric catarrh, would lead too far, as we should have to mention all the rules for diet. From the remarks on etiology, we may see that, in order to avoid gastric catarrh, the diet of some persons, as of fever patients and convalescents, but particularly of infants, must be carefully watched. In the latter case, where it is impossible to give the child the breast of the mother, or a healthy nurse, certain precautions must be exercised in the choice of cow's milk; these were mentioned under etiology: 1. The milk must be fresh; even in the city it should be brought twice daily. If it shows the least indication of acidity, it should be boiled immediately, to prevent further transformation of the sugar into lactic acid; carbonates of the alkalies may also be advantageously added to such milk, till it becomes neutral or slightly alkaline [small quantities of sulphite of soda are very good for this purpose]. 2. Milk from cows fed on oil-cake or distillery swill should not be used. In large cities, the best milk is that from brewery cows which are fed on grains. 3. The milk should be sufficiently diluted, the first three months, with about two parts of water, the second quarter with one part. 4. It should be given at regular, and not too short, intervals. During the first weeks, the bottle may be given every two hours, later every three or four hours. The shorter the intervals, the less milk should be given at each time. 5. The vessels from which the child drinks, as well as its mouth, should be carefully cleansed. Neglect of any of these rules may lead to gastric catarrh, while their observance may prove, at least, some protection for the child, against the disease.

The *causal indications* may require the administration of an emetic, where injurious or decomposing food in the stomach keeps up the catarrh. Some carry the use of emetics in gastric catarrh too far, while others neglect them too much. If we accede to the request of the patient, or, from the feeling of pressure and fulness in the epigastrium, the coated tongue and the odor from the mouth, conclude that the stomach is coated also, and, in all such cases, give an emetic of ipecacuanha or tartrate of antimony, we shall often protract the disease by letting a new injury act unnecessarily on the already diseased mucous membrane of the stomach. But just as much harm is done by the excessive fear of the injurious effects of emetics, induced by their acting also as purgatives, and by the pustular inflammation of the stomach, occasionally caused by the continued use of tartrate of antimony, but particularly by a false theory of their action. It is forgotten that the irritation of the gastric mucous membrane by the emetic, as is proved by daily experience, is not very malignant or injurious, and that the beautiful experiments of *Magendie* and *Budge* have proved that the emetic influence of ipecac. and tartrate of antimony do not result from irritation of the gastric mucous membrane, but from absorption into the blood. By injecting tartar emetic into the veins, *Magendie* proved that vomiting could be excited even where a bladder had been substituted for the stomach.

If the prominence of the epigastrium, percussion over the stomach, eructations of gases and fluids whose smell and taste are like those of the food that has been eaten, render it certain that there are decomposing substances in the stomach, and if the sufferings of the patient justify such active treatment, it will be best to give a sure emetic, such as ipecacuanha ℈j with tartrate of antimony gr. j. In the paragraph on symptoms we have shown that, even in such cases, without the use of an emetic, the undigested and decomposed ingesta may be occasionally passed from the body quickly and uninjuriously; but this is not by any means an absolute rule. The injurious substances often remain a long while in the stomach, and when they pass into the intestines cause severe and lasting disturbance. If we can rid the stomach of the substance causing a *continued* irritation and protect the bowels from its action, we should not dread the temporary irritation of the gastric mucous membrane by the emetic. If, in such a case, we do nothing, or, instead of an emetic, prescribe the popular mixture of magnesia usta, we may just as readily cause a prolongation of the attack as if we gave an emetic at the wrong time, or without sufficient cause. Moderate fever, accompanying the gastric catarrh, does not contraindicate an emetic; but if the fever is more severe, and we have the faintest suspicion of a commencing typhus, it should not be used, for typhus almost always runs a severe course, when emetics or laxatives have been used at its commencement.

The causal indications never require the use of laxatives in the treatment of simple acute gastric catarrh. It is different when the injurious ingesta have passed into the bowels and caused flatulence, colicky pains, escape of flatus, and other symptoms which are called the passage of the gastric turgescence downward. In such cases mild laxatives, such as rhubarb or compound infusion of senna, may be prescribed; if there is excess of acid, we may use a mixture of magnesia usta (℥ss to ℥viij water, a tablespoonful every hour or two), which, in these cases, acts as a mild and efficient laxative; the purgative neutral salts are less suitable.

If there be an excessive formation of acid in the stomach that seems to keep up the catarrh, whether it be caused by the transformation of the amylacea into lactic or butyric acids, or if acetic fermentation has been induced by the use of beer or wine, and if the very moderate sufferings of the patient do not justify the use of an emetic, we should give a carbonate of one of the alkalies. The most used is the bicarbonate of soda, in doses of gr. v–x, in powder or solution; if we wish to employ it in the popular form of mineral water, we should first assure ourselves that the water furnished actually contains bicarbonate of soda, and does not simply consist of carbonic acid and water.

In spite of numerous evacuations upward and downward, small amounts of the decomposing substances not unfrequently remain in the stomach. The alkalies prescribed can neutralize the acids already formed, it is true, but they cannot entirely arrest the process of decomposition and the formation of new acid products. The substances remaining in the stomach and undergoing decomposition transfer their chemical action to the fresh and unspoiled food, and render the most harmless food injurious and even dangerous for the gastric mucous membrane of children, in whom this state most frequently occurs. In such cases it is necessary to arrest the decomposition of the contents of the stomach remaining after the vomiting and purging. It is difficult to fulfil this indication, and all the skill of the physician often fails in the attempt. If we recognize the abnormal decomposition of the contents of the stomach and intestines as the most frequent cause of infantile diarrhœa, we can at least understand the unfortunate results of its treatment, which we cannot do if we regard the gastric and intestinal catarrh as the sole disease. Even outside of the body, as is well known, it is often difficult to arrest a fermentation or other decomposition that has once begun. But the means that answer for this, outside of the body, cannot always be used in it. We cannot perfectly dry the contents of the stomach or keep them at so high or so low a temperature as to arrest decomposition; and certain substances that prevent fermentation are poisonous to the organism. But, if we regard the numerous remedies (often exactly opposite in their other qualities) which physicians employ in the diarrhæa and vomiting of children, with or without clear ideas of the reasons for so doing, and which are sometimes unmistakably serviceable, we find that they are such substances as are used outside of the organism for arresting fermentation and other decompositions. The remedies most frequently given in infantile diarrhœa are carbonates of the alkalies; mineral acids, particularly muriatic; metallic salts, especially calomel and nitrate of silver; also tannin, creasote, and nux vomica. Possibly part of these, such as the nitrate of silver and tannin, have, at the same time, a favorable effect on the irritated mucous membrane of the stomach and intestines, by their astringent action on the hyperæmia. But the greater part of these remedies, especially the one most used, calomel, cannot be said to act in this way, and their effect is only to be explained by their power of arresting decomposition. If a child is suffering from a slight gastric catarrh, which only shows itself by the characteristic vomiting and the presence of undigested milk in the acid dejections, besides strict diet (of which we will hereafter speak), we should use the mildest of the above remedies, such as the carbonates of the alkalies, with small doses of rhubarb; a well-known and popular form of these is the pulv. rhei comp.; if the diarrhœa is more severe, we may give the tinc. rhei aquosa. An old and extensively used mode of giving the latter is in a mixture of tinctura rhei aquosa ʒ ij, with liq. potassii carbonici gtt. xij, aqua fœniculi ℥ ij, and syrupus simplex ʒ ij, of which a teaspoonful is to be taken several times daily. If this treatment is inefficacious, if the decomposition in the stomach continues, and the passages become more frequent, we may give small doses of calomel, a plan that has long been justly popular in the treatment of infantile diarrhœa. I usually give ⅛ to ¼ gr. two or three times daily. *Bednar*, who prefers calomel to all other remedies in this disease, gives it combined with jalap, in larger and more frequent doses. His prescription is: "℞ calomel, gr. iv; pulv. jalap, gr. ij; sacchar. alb. ʒ ss; ɱ. ft. pulv. no. viij. S. Take one powder in water every two hours." Even this treatment is not always successful. The evacuations often persist in spite of the most rigid diet and the free employment of calomel, until we fear to use any more mercury, although, from the continuance of the vomiting and purging, but little of it seems to be absorbed, and hence mercurial stomatitis rarely occurs. In such cases, every practising physician sometimes finds himself in a position where he is obliged to give up the remedy from which he has seen the best effects and which he usually trusts most, and try one in which he has less confidence. He may even feel around from one remedy to another. There are no definite and certain indications for the cases where nitrate of silver, tannin, muriatic acid, tincture of nux vomica, etc., are respectively advantageous. Usually the remedy that was efficacious in the last attack is given; if it fails, others are tried. Without laying particular stress on it, I would recommend very small doses of nitrate of silver (℞ argent. nitrat. gr. ¼; aquæ distillat. ℥ ij. ɱ. S. Take a teaspoonful every half hour or hour), and frequent potions of ice-water, in those cases where there are excessive vomiting, great thirst, and copious watery evacuations. If there be no vomiting, but great purging, and calomel does not answer the purpose, I usually give tannin (℞ tannin, ℈ ss; aquæ distillat. ℥ iij. ɱ. S. A teaspoonful every two hours). In mild but prolonged cases I give muriatic acid in mucilage. I have not much experience in the use of tincture of nux vomica, creasote, or tincture of muriate of iron.

In gastric catarrh, caused by catching cold, the causal indications demand diaphoretic treatment.

When induced by unknown epidemic influences, there are no causal indications to fulfil.

For the fulfilment of the *indications of the disease*, it is just as necessary to follow out the strictest dietetic rules, as it is unnecessary to give medicine. Experience teaches that the abnormal hyperæmia, mucous secretion, etc., of the gastric mucous membrane readily and speedily disappear on the removal of the causes which had induced or kept it up. But, as there is no doubt that even the mildest ingesta may maintain catarrhal hyperæmia, it is safest to keep patients with acute gastric catarrh without food for a while—to let them fast entirely. This is particularly advisable in the form called status gastricus. This order is often objected to; anxious mothers can hardly make up their minds to refuse their children all nourishment, even for a short time; adults with acute gastric catarrh do not feel hungry, it is true; but they have a longing for salty, piquant food. The more we insist on the fasting, the better results we shall have. If the disease is protracted, if it is accompanied by fever, or if, on account of the consumption of tissue, caused by the fever, we fear continuing the starvation, we should give nourishment in the *fluid* form, as that causes least irritation. In choosing this nourishment, we must remember that the gastric secretion is rendered alkaline from the admixture of mucus, and its digestive power greatly impaired. Hence we should usually forbid milk, eggs, and meats, which require acid gastric juice for their assimilation, and, as long as there are no signs of abnormal formation of acid, we should only permit amylaceous food. The so-called water-soups are very suitable nourishment for persons with protracted gastric catarrh.

It is exceedingly difficult to manage the diet of children with acute gastric catarrh, which has been caused, and is kept up, by decomposition of the contents of the stomach, that it is difficult to arrest. Milk, which is the most suitable and natural food for children, is injurious to them in these cases, because it quickly decomposes; then arises the difficult question: What shall we give them instead of milk? Under these circumstances, what nutriment will not be decomposed and transformed into injurious substances? We may easily satisfy ourselves that oat and barley gruel, as well as arrow-root and panada, are changed, and become sour as quickly as milk.

For the successful treatment of the disease in question, we should carefully remember that the children do not suffer from hunger, even if we withdraw all nourishment for a day or two, and feed them on fresh water alone, avoiding even the addition of sugar. If, under this treatment, the vomiting and purging cease, if the water be restored to the thickened blood, the collapse often disappears quickly, and it looks as if the fasting child were recovering, then we commence gradually with small quantities of diluted milk. If this be rejected again and again, and it appears dangerous to subject the children to a longer abstinence, I can recommend teaspoonful doses of beef-essence, which is prepared by cutting the flesh into small cubes, placing these in a bottle (without adding water), closing this securely, and leaving it in a vessel of boiling water for several hours.

The *indicatio morbi* very rarely calls for the so-called antiphlogistic remedies. Abstraction of blood, general as well as local, may be dispensed with. In severe cases, characterized by excessive vomiting and thirst, *cold* is serviceable. Both in cholera morbus and cholera infantum the use of ice-water and small pieces of ice is beneficial, as is also the application of cold compresses to the abdomen; these should be frequently renewed.

We can speak even more decidedly against the use of *muriate of ammonia* in the treatment of acute gastric catarrh than we did of its use in bronchial catarrh. We cannot depend on its anticatarrhal action, and its employment can only increase the difficulty.

*Carbonic acid* is very popular in the treatment of this disease; it is given as effervescing powder, or effervescing mixture, or as carbonic-acid water. It usually causes eructation very soon, and this appears to bring up other gases from the stomach, so that there is almost always momentary relief. It is not claimed, however, that carbonic acid, which everywhere acts as an irritant, moderates the hyperæmia of the stomach, and has any direct influence on the rapid cure of the disease.

It is different with the use of the *carbonates* of the alkalies; they lessen the toughness of the secreted mucus, and facilitate its evacuation; hence, independent of their use for fulfilling the causal indications (see above), they deserve full consideration in the later stages of acute gastric catarrh. Moreover, the alkaline carbonates appear to assist the secretion of the gastric juice; at least *Blondlot* and *Frerichs* observed that, after giving carbonates of the alkalies, enough acid gastric juice was formed, not only to neutralize the alkali, but to give the contents of the stomach an acid reaction. In the status gastricus they are usually given in the form of soda-water, or tinctura rhei aquosa.

Further rules are rarely required for the treatment of *symptoms.* Among the symptoms that most frequently call for treatment is *vomiting*, and, where the bowels are affected at the same time, *diarrhœa.* If moderate, these may be regarded as favorable symptoms, and require no special treatment; but sometimes, as in cholera morbus, or cholera infantum, they may be so severe that the blood will be much thickened by the loss of water, and life be endangered. Opium is the most usual prescription for the excessive vomiting and purging. We do not know exactly how opium arrests these symptoms. If it only paralyzed the intestines, and so diminished the number of the stools without decreasing the secretion of the mucous membrane, it would be of little real benefit; but it really seems as if, besides the influence it has on the movements of the intestines, and perhaps as a direct result of this, it also limited the secretion of the intestinal mucous membrane. Hence, if, in cholera morbus, ice-water do not arrest vomiting, and the passages become more numerous, we should give gr. ss of opium in powder, or its equivalent of laudanum, alone or with analeptics. In spite of our dislike to give opium to children, and in spite of our belief that it answers neither the *indicatio morbi*, nor the causal indications, we may be obliged to give small doses of it in cholera infantum. In cholera morbus, or cholera infantum, the greater the collapse, the weaker the pulse, and the lower the temperature, the more necessary it becomes to use stimulants; inwardly we may give small doses of wine, ether, coffee; outwardly we may use sinapisms.

On the other hand, in the course of acute gastric catarrh, in spite of the alkalies that have been exhibited, a quantity of mucus may collect as a product of the disease, and by its decomposition cause an obstinate continuance of the affection, or, after this has run its course, may retard convalescence and disturb digestion. If, in the later stages of gastric catarrh, the painful attacks of vomiting, which, from time to time, throw out quantities of mucus, the loss of appetite, or the slow recovery, render it probable that such a state of affairs exists in the stomach, it may be necessary to give an emetic.

## CHAPTER II.

### CHRONIC GASTRIC CATARRH.

ETIOLOGY.—Chronic gastric catarrh sometimes occurs as a result of the acute affection; when this is protracted or relapses frequently, sometimes it originates as a chronic disease. Hence the etiology is mostly the same as that of acute gastric catarrh. It may be caused:

1. By all injurious influences that excite the above disease, when they act continuously or repeatedly. But the habitual misuse of spirituous liquors deserves particular mention, as it is by far the most frequent cause of chronic gastric catarrh. We also ob-

serve that alcohol acts the more injuriously the more undiluted it is taken; hence brandy-drinkers are most liable to the affection.

2. In many cases chronic gastric catarrh depends on congestion of the gastric mucous membrane. The obstruction of the circulation inducing this congestion may be located in the portal vein; hence we find that all affections of the liver, by which the portal vein or its branches are compressed, are always accompanied by chronic gastric catarrh. But more frequently the obstruction lies beyond the liver; all affections of the heart, lungs, or pleura, that cause an overfilling of the heart and obstruction of the vena cava, also obstruct the escape of blood from the liver, and hence from the stomach; consequently, in emphysema, cirrhosis of the lungs, valvular disease of the heart, etc., we meet chronic gastric catarrh just as often as we do cyanosis of the skin, and both affections must be induced in the same way.

3. Chronic gastric catarrh often accompanies phthisis and other chronic diseases. In part I., we showed that patients with incipient phthisis often complain more of their gastric catarrh than of the lung trouble, and that is what first induces them to apply for aid.

4. It always accompanies cancerous or other degeneration of the stomach.

ANATOMICAL APPEARANCES.—In chronic gastric catarrh, the mucous membrane is often reddish brown or slate gray, just as it is elsewhere when it is the seat of chronic catarrh. This is caused by small capillary hæmorrhages in the tissue of the mucous membrane, and the transformation of the hæmatin into pigment. Instead of the fine injection seen in acute catarrh, we usually find a coarse anastomosis, and in some places dilatations of the vessels. Moreover, the mucous membrane has become hypertrophied, it is thicker and firmer, and, even when the muscles are not contracted by rigor mortis, we find the mucous membrane forming numerous folds, and some parts of it are occasionally elevated to soft spongy nodules by a velvety hypertrophy. We often find innumerable small prominences, separated by superficial furrows, a state described as the *état mamelonné*. The mammillated appearance most frequently depends on partial hypertrophy of the gastric mucous membrane, by which some of the glands and their insterstitial connective tissue have been enlarged. *Frerichs* asserts that it is also caused by roundish collections of fat in the submucous tissue, or by the development of closely-crowded closed follicles; according to *Budd*, they result in some cases from overfilling of the gastric glands with retained secretion. These changes are found most frequently and farthest advanced in the pyloric end of the stomach. The inner surface of the stomach is covered with a grayish-white, tough mucus, which clings firmly to it.

The thickening is not always limited to the mucous membrane; sometimes the submucous and muscular tissues are changed to a fatty mass, several lines or even half an inch thick. This thickening of the wall of the stomach also depends on simple hypertrophy, in which there is both a new formation of muscular cells, and an increase of the submucous and intermuscular connective tissue. On the cut surface the thickened muscular tissue shows a pale grayish-red, soft, fleshy mass, traversed by parallel connective-tissue striæ, running from without inward, and having a peculiar fan-like appearance. Occasionally the whole pyloric end of the stomach, and especially the pylorus itself, is changed in this way; in other cases the thickening of the walls of the stomach is more circumscribed, and forms certain prominent nodules (*Foerster*). The pylorus may be greatly constricted by thickening of the walls of the stomach from simple hypertrophy, and this constriction may cause great dilatation of the part of the stomach above the stricture.

SYMPTOMS AND COURSE.—In chronic gastric catarrh, the patients complain most of a disagreeable feeling of pressure and fulness in the stomach, which is increased by eating, but rarely amounts to severe pain. Where the latter occurs after eating, and the epigastrium is sensitive to pressure, we must always suspect that there is not simply chronic gastric catarrh, but that it is complicated by some more serious disease. With the feeling of fulness there is almost always a prominence of the epigastrium, caused by the filling of the stomach with gas, and by the ingesta remaining in it for a long while. The gases in the stomach are formed in chronic catarrh also by the decomposition that the ingesta undergo when the gastric juice, which has become alkaline, no longer causes normal digestion, and the mucus in the stomach acts as a ferment on its contents. The abnormal decomposition is assisted, however, by the fact that, although the muscular coat of the stomach has increased in thickness, its functions are paralyzed by serous infiltration. When the movements of the stomach are retarded, food remains in it a great while, and undergoes abnormal decomposition. From time to time there is eructation of gases having the same composition as those formed in acute gastric catarrh. With the eructation, which is a constant symptom of chronic gastric catarrh, besides the gases, small quantities of sour or rancid fluid often rise into the mouth, and are either spit out or swallowed again. The formation of lactic and butyric acids from the transformation of the amylacea is often very extensive, and the sour and acrid fluids, rising into the œsophagus and pharynx on belching, cause the burning feeling called *heartburn*.

Occasionally, beside the above symptoms there is *vomiting;* this, however, is not constant; on the contrary, it is rather rare. According to the observations of *Frerichs*, to whom we owe most of what we know concerning the anomalies of digestion, the hydrocarbons are occasionally changed into a tough filamentous mass resembling gum, and which is not unfrequently formed by lactic-acid fermentation outside of the body. The vomited substances not unfrequently consist of large quantities of this non-nitrogenous material, which is thrown up in mucous filamentous masses after painful retching. In other cases, pure mucus with an insipid fluid is thrown up; this form of vomiting occurs chiefly in the chronic catarrh of drunkards, and constitutes the celebrated *vomitus matutinus* (water-brash). *Frerichs*, who has carefully examined these masses, found that they were usually alkaline, had a low specific gravity, always contained sulphurets, and that alcohol added in excess threw down a white flocculent precipitate which rapidly converted starch into sugar. This peculiarity of the fluid showed that it was not formed in the stomach but in the salivary glands. We have before said that irritations and diseases of the stomach increased the salivary secretion; hence it appears that in drunkard's chronic gastric catarrh, the saliva swallowed during the night is thrown off in the morning as *vomitus matutinus*. In simple, non-complicated chronic gastric catarrh, unaltered food is very rarely vomited. If this does occur, it is usually mixed with a quantity of mucus, and from admixture of butyric acid has a disagreeable, acrid smell and taste, and occasionally contains a peculiar microscopic formation, the so-called *sarcina ventriculi*. It can scarcely be doubted that the sarcina, which, when it occurs in the stomach, is always found in great numbers, is an algoid growth. It presents cells of the $\frac{1}{400}$ to $\frac{1}{300}$ of a line in diameter, with square surfaces divided into four regular parts; usually several, sometimes very many of these, are united into smaller or larger squares. It is not to be supposed that it is this parasitic plant which, acting as a ferment, causes an abnormal decomposition of the contents of the stomach, for, in healthy stomachs (though it rarely occurs there, it is true), its presence does not induce this abnormal decomposition.

The *sensation of hunger* is almost lost, even when the patient is much emaciated, and the body is very much in need of support; frequently the patients can hardly be persuaded to take nourishment. In other cases there is a feeling of hunger, but even a few mouthfuls satisfy it. Finally, in some cases, particularly where much acid is formed, there is occasionally pain in the stomach, accompanied by faintness. As this is generally relieved by eating, it is commonly called "wolfish appetite" (heiss-hunger). As there is no fever, the thirst is not increased; it is often less, like the appetite.

If the chronic catarrh extends from the stomach to the mouth, we have, at the same time, the symptoms of chronic oral catarrh: the tongue is coated, shows the impression of the teeth along its sides; there is a stale, slimy taste, and a more or less fetid smell from the mouth. But a clean tongue and absence of the other symptoms of oral catarrh do not at all prove that the stomach is healthy.

Not unfrequently the chronic gastric catarrh extends to the intestines, and, besides the symptoms above described, we have those of chronic intestinal catarrh. We must, however, bear in mind that every intestinal catarrh does not cause diarrhœa, because it is not always accompanied by fluid secretions, or large quantities of mucus. There is more apt to be somewhat obstinate constipation, because the movements of the intestines, like those of the stomach in chronic gastric catarrh, are greatly impeded. Decomposition of the contents, which thus remain a long time in the intestines, continues; there is flatulence, which renders the belly tense, and the patients, who feel relieved by the escape of flatus, usually ascribe their difficulty to the "movements of the flatus."

Occasionally, also, the catarrh extends from the duodenum to the ductus choledochus, and there are retention and absorption of bile. We shall find that the jaundice caused by gastro-duodenal catarrh is the most frequent form of icterus.

In regard to the *general state* of the patient, the severe headache, pain and weakness of the limbs, and other general symptoms which accompany acute gastric catarrh, are usually absent in the chronic form; but, on the other hand, there is usually some *mental depression*. If this state be designated as hypochondria, because the abnormal excitement of the brain depends on abnormal conditions of the abdominal viscera, there can be no objection to it; but the mental disturbance accompanying gastro-intestinal catarrh should not be distinguished from other forms of melancholia by the fact that the bodily state is the sole object of the gloomy thoughts. I have seen a *general* discouragement, an under-valuation of mental power, despair as to business, etc., induced by chronic gastric catarrh, and have seen these symptoms disappear on the cure of the disease. Only a few years since I treated a very wealthy man for chronic gastric and intestinal catarrh, who, during the disease, thought he was near bankruptcy, and left unfinished a building that he had begun, because he thought he had not sufficient money to continue it. After spending four weeks at Carlsbad, his old strength and feelings returned, he finished his house with great splendor, and has been well ever since.

When the disease lasts a long while, the *nutrition* of the patient suffers from the disturbance of chymification, as well as from the interference with resorption, caused by the tough mucus on the gastric and intestinal mucous membrane; the fat disappears, the muscles become relaxed, and the skin dry. Not unfrequently, scorbutic affections, loosening of the gums, bleeding from them, and even ecchymoses on the extremities, are seen. Excessive emaciation is suspicious; when it occurs, we may fear that the gastric catarrh is a secondary or symptomatic affection, which is caused or maintained by carcinoma.

The frequent *change* observed in the urine in this disease is peculiar, and difficult to understand. Even taking into consideration the fact that disturbed absorption must excite a change in the excretions, we cannot explain the high color, the sediments of urates, or the frequent appearance of quantities of oxalate of lime in the urine of patients who have chronic gastric catarrh (see chapter on dyspepsia).

As to the *course and results* of chronic gastric catarrh, the symptoms above described may run on for months, or even years, with more or less severity, and often with frequent variations of intensity. When the causes can be removed by proper treatment, the disease is often cured; in other, not very frequent, cases, it induces severer lesions of the stomach, particularly *chronic ulcer of the stomach* (?), and, when induced by mechanical disturbances, it may cause *hæmorrhage* from the stomach. Not counting the secondary affections, this disease is rarely fatal; although cases do occur where the patients finally die of marasmus and dropsy, but they more frequently die of the diseases complicating or causing the gastric catarrh.

Hypertrophy of the membranes of the stomach cannot be recognized during life, unless the calibre of the pylorus is diminished. This may result from the villous hypertrophy of the gastric mucous membrane which we described among the anatomical appearances.

Stricture of the pylorus, from hypertrophy of the mucous membrane, impedes the exit of the contents of the stomach; a new cause of abnormal decomposition is thus added to those resulting from the catarrh. This explains why, in stricture of the pylorus, the symptoms that we deduced from abnormal decomposition of the contents of the stomach (such as eructation of gases and badly-tasting fluids, heartburn, etc.) reach even a higher grade, and are more distressing than in simple chronic gastric catarrh. Besides this, we have *vomiting*, which does not occur, or comes only occasionally, in many, or even in most cases of simple chronic gastric catarrh, as one of the most constant symptoms of pyloric obstruction; it usually comes quite regularly two or three hours after eating. This is occasionally different when the stomach is much distended, and hence can hold a large quantity; then there may be no vomiting for two or three days; after such pauses, enormous quantities are evacuated at one time. In such cases there may be a certain regularity.

In stricture of the pylorus, the vomited masses almost always consist of more or less digested food embedded in mucus, which smell disagreeably sour and rancid; they usually contain quantities of lactic and butyric acids, and frequently sarcina. If there be decided acidity which cannot be checked, with frequent and regular vomiting, there is very probably pyloric obstruction; the diagnosis becomes more certain if we can make out a consecutive dilatation of the stomach (which may become large enough to fill the greater part of the abdomen); this may sometimes be done by inspection of the abdomen, when the distended stomach may be seen as a convex prominence, extending down to the navel, or even below it. *Bamberger* calls attention to the fact that where the stomach is very low down, not only the greater, but also the lesser curvature, may be made out as a prominence extending from the cartilages of the false ribs on one side to those on the other, just below the so-called "pit of the stomach," which is sunk in. On moving the skin in the epigastrium, we occasionally observe the region of the stomach to swell up and form a tense tumor. This appearance, along with which the contours of the stomach may be felt, is doubtless due to the tension of the organ over its fluid and gaseous contents which cannot escape, and to its consequent change from its usual relaxed state to a more spherical shape. The most elevated segments of this sphere become visible on the abdomen, while those lying deeper are perceived only by the touch. This change of the stomach from a relaxed, loose bag, to an elastic, tense, spherical bladder, is usually accompanied by a disagreeable and more or less painful sensation. Apart from the transitory symptom just described, we notice the slight resistance of the epigastric region, which *Bamberger* has so well described as feeling like an air-cushion. The prominence of the epigastrium decreases or disappears when the patient has vomited freely. In one case, treated at the Greifswalder clinic, on giving the patient an effervescing powder, the region over the stomach, and as far down as the navel, swelled considerably, and the contours of the stomach were clearly marked. Then, if part of the carbonic acid were belched up, the swelling subsided. When the stomach is full of food, the percussion dulness is very extensive; but if, as is usually the case, it contains a quantity of gas at the same time, the percussion-sound is particularly full and tympanitic at the prominent places. If the patient changes his position, the solid substances always go to the lower parts of the stomach, and the bounds of the dull and clear percussion change.

The above symptoms render the existence of pyloric constriction very probable, but we can only ascribe this to simple hypertrophy of the walls of the stomach, when we can exclude the other and more frequent forms of stricture, particularly the cancerous and the cicatricial stricture, not unfrequently left after the healing of a chronic ulcer.

The *prognosis* of chronic gastric catarrh agrees with what we have said of its course. Stricture of the pylorus must be classed among the frequently fatal diseases, for patients with this disease always die, sooner or later, of marasmus or dropsy.

TREATMENT.—Of all serious chronic diseases, chronic gastric catarrh probably gives the best result from rational treatment.

As we have described, in the first part of this chapter, the injuries which, according to the duration of their action, induce acute or chronic gastric catarrh, we may, in speaking of the causal indications for treatment, refer to that description, and we have little to add to it. These indications are rarely fulfilled by the use of an emetic, as there are rarely any injurious substances in the stomach that can be considered as keeping up the disease. On this point we often meet opposition. It is difficult to convince the patients that the pressure they feel is not excited by "something heavy on the stomach," and that an emetic would bring no relief, but rather would make matters worse. The causal indications urgently require the *forbidding of all spirituous liquors*, if their continued use has caused, and is keeping up, the affection. This command will rarely be obeyed; nevertheless, we must not weary of repeating it. Temperance lecturers, who also demonstrate the terrifying results of brandy-drinking on the stomachs of topers, usually preach to deaf ears, it is true, but they attain some undeniable results, and these should encourage the physician to persist in his advice. In the chronic catarrh caused by repeatedly catching cold, or by the action of a moist, cold climate, the indication is to excite the activity of the skin by warm clothes, warm baths, and similar means. Such cases are not at all rare; and, even at Greifswald, patients who have come here without preparing for the damp, windy climate, by dressing more warmly, are often affected with chronic gastric catarrh, which is better in summer, worse in winter, and is not cured till the causal indications are properly attended to. When the disease results from congestion, the causal indications can rarely be fulfilled.

Dietetic rules are also of the greatest importance in fulfilling the *indications of the disease*. It is not possible to keep the patients fasting throughout this tedious complaint, but we should most carefully select their food, and urgently insist on its exclusive use. The more precise the rules, the more carefully they will be followed, and, if the prescribed diet be considered as a regular treatment, it is usually observed by the patient with painful conscientiousness. Since the use of meat, and other animal food, particularly requires activity of the stomach, one might suppose that the indication was to allow only vegetable diet to a patient with chronic catarrh of the stomach, the digestive power of whose gastric juice has become weakened, but experience teaches the contrary. The power of the gastric juice to convert the protein substances into peptone (*Lehman*), or albuminose (*Mialhe*), is diminished in chronic catarrh, it is true, but it is not entirely lost. If they be given judiciously and in proper form, the patients improve more than if fed only on amylacea, from which quantities of lactic and butyric acids are formed in the stomach. From what has been said above, it follows, of course, that fat meat and sauces are to be forbidden; that the food is to be carefully chewed, and only small portions of it swallowed at a time. Some patients get along very well when they only eat concentrated, unskimmed meat broth; others do so when they only eat cold meat, and but little white bread. The latter prescription is especially useful in patients who suffer from excessive acidity, and, in very obstinate cases of this kind, instead of the "cold-meat treatment," we may recommend the use of salt or smoked meat. If it be considered curious that some patients bear meat better when in this indigestible form than otherwise, it is because the fact is overlooked that smoked and salt meat, even if indigestible, has this advantage over fresh meat, that it is not so readily decomposed as fresh meat. In one case that I treated, the patient, who had chronic gastric catarrh, with great inclination to acidity, knew exactly when he must abandon all other food (because it increased the gastric juice), and limit himself to the use of lean smoked ham, sea-biscuit, and a little Hungarian wine. The exclusive use of milk, the so-called milk-cure, agrees wonderfully with some patients, while others cannot stand it at all, and we cannot certainly tell beforehand which will be the case. Butter-milk suits many patients better than fresh milk. In *Krukenburg's* clinic I have seen very brilliant results from the prescription, "when the patient is hungry, let him eat butter-milk; when he is thirsty, let him drink butter-milk." Perhaps fresh milk is not so well borne, because it readily curdles in the stomach, and forms large, firm lumps, while in the butter-milk the casein is already curdled, but finely divided.

Dietetic treatment does not succeed so often in chronic as in acute gastric catarrh, but we have some very efficient remedies for the former disease. The chief among these are the alkaline carbonates. We have already recommended bicarbonate of soda, in divided doses, and tinctura rhei aquosa, in prolonged attacks of acute gastric catarrh.

Where chronic gastric catarrh is obstinate, we should try soda water, or the natural soda waters of Ems, Salzbrun, Selters, and Bilin, as well as the waters which, besides carbonate of soda, contain sulphates of the alkalies and earths or chloride of sodium. The use of the waters of Karlsbad and Marienbad has the most wonderful results. The highest recommendation that could be given for them is the fact that they are recommended by parties whom no one can accuse of being easily deceived by therapeutic results: the learned professors of the Vienna and Prague schools prize the use of the warm springs of Karlsbad as the best remedy for chronic gastric catarrh, and even for chronic ulcer of the stomach. Moreover, the numerous cases where obstinate jaundice was cured by the use of the waters of Karlsbad were almost always those where it was due to gastro-duodenal catarrh. There is no reason to delay this treatment until the catarrh of the stomach and duodenum has caused jaundice, or to suppose that it will be less efficacious if this complication be wanting. If the circumstances of the patient permit, the treatment may be followed out at Karlsbad or Marienbad; at these places the anecdotes of the frightful results from errors of diet during the use of the waters so terrify the patients that the diet required by chronic gastric catarrh will be certainly adhered to while there. Even after returning home, the patients subject themselves to the strictest regimen for months, fearing that the waters may revenge themselves even yet for the slightest errors of diet. If obliged to use the waters at home, it makes little difference from which of the Karlsbad springs they come, as they vary little except in their temperature, and they may be warmed to any desired extent. In Karlsbad the springs of lower temperature, as the Schlossbrunnen and Theresienbrunnen, are most frequently used in chronic gastric catarrh. If there be no coincident obstinate constipation, soda water will often succeed quite as well, provided it be properly used, i. e., if the patient diets the same as at Karlsbad. After eating but little, and not very late, the night previous, he must drink the soda water in the morning while fasting, and must not breakfast for an hour after the last glass of water, so that the medicament may not be mixed with the ingesta, but may act undiluted on the gastric mucous membrane, and on the mucus covering it. The results from this treatment are the most brilliant that are ever attained in medicine.

The ter-nitrate of bismuth and the nitrate of silver have a great reputation in the treatment of chronic gastric catarrh. These metallic salts may be beneficial, both by arresting decomposition in the stomach and by their great astringent action on the hyperæmic and relaxed mucous membrane. I have used these remedies in my clinic in very large doses (bismuth nitrat. gr. x, argenti nitrat. gr. j—ij, at once), given like the alkaline carbonates, on an empty stomach, before breakfast. Most patients bore these doses very well; severe pain, nausea, or vomiting never occurred, and there was diarrhœa in only a few cases. But the results were very varied; while in some cases there was very rapid improvement, in others there was none, and I was unable to find any cause for difference between them.

In chronic gastric catarrh we sometimes dare not continue the mild diet; on the contrary, slightly-seasoned and salty food is much better borne than unseasoned and unirritating. When this state of "atony of the gastric mucous membrane" occurs, we should carefully prescribe preparations of iron and mild stimulants. The Eger Franzbrunnen, and even the chalybeate waters of Pyrmont, Driburg, or Cudowa, are better borne, and do more good than those of Karlsbad and Marienbad. When the mucous membrane is in this state, the best remedy is, ipecacuanha, gr. ss—j, pulv. rhei, gr. iij—iv, in pill, to be taken before meals, as recommended by *Budd*. The tinctura rhei vinosa, Hoffmann's visceral elixir, ginger, calamus, etc., do good in these cases; but we must beware of going too far in the use of these remedies, or of giving them in improper cases, or too large doses.

The symptoms rarely require the application of leeches or cups to the epigastrium; they are only to be used when there is great pain. Difficult as it is to understand, the pain is almost always relieved by the abstraction of blood. In those cases where the hyperæmia and catarrh of the stomach are symptomatic of great abdominal plethora, depending on compression of the portal vein, or obstruction to the flow of blood from the hepatic veins, surprising results are often obtained by an abstraction of blood from the anastomoses of the portal vein by applying leeches at the anus. Narcotics, which are almost indispensable in treating ulcers of the stomach, are rarely required in chronic gastric catarrh. Emetics may be employed under the circumstances in which they were advised in acute gastric catarrh, but we must be more careful with them, as we do not know that ulceration may not have occurred already. The constipation which almost always exists is to be treated by enemata or laxatives; the medicines most used are rhubarb and aloes, and, in obstinate cases, extract of colocynth. Several of these articles are usually combined; the officinal (in Germany) and much-used compound extract of rhubarb contains aloes, rhubarb, and jalap. *Budd* says, also, that aloes and colocynth act chiefly on the rectum, and irritate the stomach but little, so that they are the best purgatives in chronic gastric catarrh; he warns against the use of senna and castor-oil.

## CHAPTER III.

### CROUPOUS AND DIPHTHERITIC INFLAMMATION OF THE GASTRIC MUCOUS MEMBRANE.

Croupous and diphtheritic inflammation of the gastric mucous membrane is rarely observed, unless poisonous substances have acted on it (see Chapter V.). In some cases, in infants, the catarrhal form of inflammation increases to the croupous; in others, croupous and diphtheritic gastritis belongs to the secondary inflammations occurring in the acute infectious diseases, especially in typhus, septicæmia, and small-pox.

Croup membranes rarely spread over a great extent of the gastric mucous membrane; they are usually limited to small circumscribed spots. The diphtheritic sloughs also form isolated patches; on falling off, they leave losses of substance with discolored ragged bases.

Unless pseudomembranes are vomited up, the disease is rarely, if ever, recognized during life. The difficulties the disease causes in children can never be rightly interpreted, and the severe symptoms of septicæmia, typhus, etc., are so little modified by an intercurrent croupous or diphtheritic gastritis, that in such cases also diagnosis is impossible.

## CHAPTER IV.

### INFLAMMATION OF THE SUBMUCOUS CONNECTIVE TISSUE—GASTRITIS PHLEGMONOSA.

Inflammation of the submucous connective tissue, which *Rokitansky* compares to pseudoerysipelas, is also rare. It occurs either as a primary affection, without perceptible cause in previously healthy persons, or, like the above, it is a so-called secondary or metastatic inflammation, and, as such, accompanies typhus, septicæmia, and similar diseases.

The submucous tissue of the stomach is diffusely infiltrated with pus, which collects in its distended meshes; more rarely there are circumscribed abscesses in the submucous connective tissue. The undermined mucous membrane is thinned, and subsequently it has numerous small openings, from which the pus trickles out as through a sieve. The inflammation soon extends to the muscular layer, the submucous tissue, and peritonæum. If the patient recovers, cicatricial tissue may form in the meshes of the submucous, and strictures may thus result, as is shown by specimens in the Erlangen Museum.

The most important symptoms of the disease are severe pain in the epigastrium, vomiting, great anxiety, high fever; later, there are symptoms of peritonitis, the patient collapses, and usually dies in a few days. Of course, a diagnosis can only be certainly made in a few cases, where, with the above symptoms and vomiting of pus, we are able to exclude other forms of gastritis, particularly those caused by poisons. The treatment can only be symptomatic.

## CHAPTER V.

### INFLAMMATIONS AND OTHER CHANGES IN THE STOMACH FROM CAUSTICS AND POISONS.

ETIOLOGY.—The changes in the stomach caused by the action of concentrated acids, caustic alkaliès, and some metallic salts, depend on the fact that these substances unite chemically with the tissue of the walls of the stomach, whose organic structure is consequently destroyed. The changes that vegetable or animal poisons excite in the gastric mucous membrane, on the contrary, cannot be traced to chemical processes.

Poisoning by carelessness is most frequently induced by copper-salts, sulphuric acid, or vegetable poisons being taken into the stomach; while intentional poisoning occurs most frequently from arsenic or sulphuric acid.

ANATOMICAL APPEARANCES.—If dilute mineral acids have acted on the mucous membrane, only the epithelial and superficial mucous layers are changed to a soft, brownish or black slough. If a quantity of concentrated acid has reached the stomach, all the layers of the mucous membrane are converted into a soft black mass, which may become several lines thick from imbibition with bloody watery fluid. The muscular tissue becomes softened or gelatinous, and very friable; more rarely both it and the serous membrane are entirely decomposed, and the stomach perforated. These changes are usually limited to a few longitudinal folds of the mucous membrane, running from the cardiac end toward the pylorus, while the rest of the membrane is reddened by hyperæmia and ecchymosis, and swollen by serous infiltration; the blood in the vessels of the stomach, and often even in the neighboring large vascular trunks, is transformed into a black, smeary, tar-like substance. Only the milder cases recover, for the parts destroyed slough off, and the loss of tissue is replaced by callous cicatricial substance. The caustic alkalies change the epithelium and the superficial, or even the deeper layers of the mucous membrane, into a pulpy, discolored mass. In these cases, more frequently than in cases of poisoning from acids, the destruction extends to the muscular and serous tissues, and so leads to perforation. When the destruction is superficial, cure may result even in such cases, after the sloughing of the necrosed parts.

Brown or black sloughs are formed by the action of corrosive sublimate, copper, or other metallic salts; these are surrounded by active injection and serous swelling of the mucous membrane. Phosphorus excites similar changes.

If gastritis occurs after poisoning from arsenic, we find one or more spots of the mucous membrane covered with a powdered, white substance, swollen, reddened, and softened to a pulp, or transformed to a yellowish or greenish-brown slough. From these sloughs extend reddened folds of mucous membrane, between which the walls of the stomach are often unaltered.

After the action of ethereal oils, or acrid vegetable or animal poisons, the remains of severe catarrhal, croupous, or diphtheritic inflammation are seen.

Symptoms and Course.—Gastritis from poisoning is peculiar, because, even where the poison used has no directly paralyzing effect on the nervous system, besides the local symptoms, there is a general depression, and particularly an almost complete arrest of the circulation. These paralytic symptoms are also seen in other severe injuries of the stomach or other abdominal viscera, but especially in perforation of the stomach from ulceration.

If a previously healthy person be suddenly attacked with severe pain, which spreads from the epigastrium over the abdomen; if this be accompanied by vomiting of mucus or bloody mucus; if there be also purging of mucus and blood, preceded by severe colicky pains and tenesmus, and the patient be at the same time collapsed, and his features distorted, his extremities cool, pulse small, and skin covered with cold, clammy sweat; there is strong ground for suspecting the action of a corrosive substance or some other poison on the gastric mucous membrane. If concentrated acids or strong alkalies have been taken, there are almost always characteristic sloughs about the mouth; the oral mucous membrane is destroyed in some places; there are severe pains in the mouth and throat; swallowing is very difficult, or impossible. After taking the metallic salts or arsenic in a diluted form, the signs of corrosion of the mouth and throat do not appear, and the symptoms of gastritis do not occur for some time. The symptoms observed in the different organs, but particularly the examination of the evacuations, show what kind of poison has been taken. In the most severe cases there is nausea, but the paralyzed stomach cannot evacuate its contents; an icy coldness spreads over the body, the paralysis becomes total, and the patient may die in a few hours. In milder cases death does not occur till later, and, when a quantity of the poison has been vomited, the symptoms of paralysis may gradually disappear, and the circulation may be reëstablished; but convalescence is usually very slow, and the patient often suffers for life from strictures in the œsophagus or stomach, or else because the poison taken has undermined the constitution in some other way.

TREATMENT.—The antidotes given in books on toxicology can only be given in recent cases, that is, within a few hours after acids, caustic alkalies, or metallic salts have been taken. If these substances have already been vomited, or have already united with the elements of the gastric mucous membrane, antidotes can do no possible good, and may prove injurious by exciting new irritation in the inflamed gastric mucous membrane. It is different with arsenic and the acrid vegetable and animal poisons, whose action continues longer, and for which the customary antidotes may be used for a longer time after they have been taken. If there be no vomiting, or if this do not suffice to rid the stomach of the poison, we may give an emetic of ipecacuanha. Besides these rules for fulfilling the causal indication, the indications from the disease itself are to use cold, as blood-letting does little or no good. We may cover the abdomen with cold compresses, which are to be frequently changed, and give small quantities of ice-water; or, if the patient can swallow, let him have small pieces of ice. For further treatment, we refer the reader to works on toxicology.

## CHAPTER VI.

### CHRONIC (ROUND, PERFORATING) ULCER OF THE STOMACH—ULCUS VENTRICULI CHRONICUM (ROTUNDUM, PERFORANS).

ETIOLOGY.—Perforating ulcer of the stomach is probably always acute; even its extension appears to be due to an acute process of destruction at its periphery and base. However, as the ulcer in question often gives the patient great trouble for years, it may rightly keep its name of "chronic ulcer." The sharp borders of the round ulcer, the absence of signs of inflammation or suppuration at its periphery, the direct observation of very recent cases, as well as the striking results of a series of experiments on animals, prove, beyond doubt, that the destruction of the wall of the stomach is not due to a gradual breaking down from suppuration, but to the formation of a slough, to a partial necrosis, and that this usually, if not always, depends on an obstruction of the blood-vessels running in the walls of the stomach and nourishing it. The death of a circumscribed portion of the wall of the stomach from cutting off its nourishment is analogous to the localized softening of the brain, infarction of the lungs, spontaneous gangrene of the toes, caused by cutting off the circulation. In the above-mentioned experiments on animals, the obstruction of the gastric vessels was induced by introduction of emboli. This mode of development of the round ulcer is rare in man, but there are some cases where certainly it has been observed. (I myself have seen a most exquisite example of it within a few years.) The obstructing clots usually form at the very site of the ulcer, and their formation seems to depend on disease of the walls of the vessel. The gastric juice quickly causes softening and entire dissolution of the dead portion of the wall of the stomach, which cannot withstand its action, so that we rarely have the opportunity of seeing the first stage of the process on *post-mortem* examination. The predisposition to chronic ulcer of the stomach is very extended. *Jaksch* and others have given us statistics of its frequency at different ages, in different sexes, and in different employments, etc. In the accounts of two thousand three hundred and thirty *post-mortem* examinations, *Jaksch* found round ulcers mentioned fifty-seven times, and cicatrices fifty-six times; so that, to about every twenty bodies, there was either an ulcer or a cicatrix. *Willigan*, *Brinton*, and others came to similar conclusions. Round ulcer is rarely found in children, but, on the other hand, it is quite frequent about puberty. Females are much more disposed to it than males. I think there is no doubt that poverty of the blood and chlorosis, those frequent results of sexual disturbances, have great influence in causing the round ulcer, and that they do so because abnormal states of the blood induce diseases of the walls of the vessels, and hence favor the formation of thrombi. In other cases acute or chronic catarrh of the gastric mucous membrane appears to cause disease of the walls of the vessels, and consequently thrombosis.

The exciting causes of round ulcer are entirely unknown. We cannot deny the possibility of its being induced by the injuries usually named; such as the use of very hot or very cold food and drink, the misuse of liquor, and other errors of diet. But it is very remarkable that, in spite of the frequency of chronic gastric catarrh in topers, they rarely have the round ulcer.

Anatomical Appearances.—The ulcer which we are considering occurs almost exclusively in the stomach or upper part of the duodenum, while it is only rarely seen in other parts of the intestinal canal. It is most frequently situated in the pyloric portion of the stomach, oftener in the posterior than in the anterior wall; and almost always at the small curvature or its vicinity; it is rarely seen at the fundus. Usually these is only one ulcer, occasionally two or more, and not unfrequently a recent ulcer near the cicatrices of some that have healed. In typical cases, according to *Rokitansky's* classical description, there is a circular hole with sharp borders in the serous coat of the stomach, as if a piece had been cut out with a punch. Regarded from within, the loss of substance is greater in the mucous membrane than in the muscular coat, and greater in this than in the serous coat, so that the ulcer is in terraces and looks like a shallow funnel. The ulcers vary from $\frac{1}{4}$—$\frac{1}{2}$ inch in diameter; old ulcers attain the size of a thaler or the palm of the hand. At first they are round, after they have existed some time they become elliptical, or bulge out in some places, and so become irregular. They spread transversely in the course of the vessels, so that the stomach is occasionally surrounded by a girdle as it were.

Sometimes the ulcer heals before it has perforated all the coats of the stomach. If the loss of substance has been limited to the mucous and submucous tissue it is replaced by granulations; these are transformed to shrinking cicatricial tissue; they draw the edges of the ulcer together, and a stellate cicatrix of variable size forms on the inner surface of the stomach. If the ulcer has penetrated deeper and destroyed the muscular coat also, when it heals up, the cicatricial contraction of the neoplastic connective tissue will contract the peritonæum also into a stellate figure; its inner surface may even be retracted into the form of a fold in the stomach. If the ulcer were very large, its healing may cause a stricture, as the diameter of the stomach will be much diminished by the cicatricial contraction; this will remain as an incurable obstacle to the passage of the contents of the stomach into the bowels.

If the ulcer be located in the small curvature, as is usually the case, even if all the walls of the stomach be destroyed, escape of the contents into the peritoneal cavity may be temporarily or permanently prevented. For, while the ulceration progresses outwardly, local peritonitis occurs at the affected part; the threatened portion of serous membrane becomes attached to the neighboring organs; if it then be destroyed, these organs (most frequently the pancreas, the left lobe of the liver or the omentum), which are firmly attached to the edges of the ulcer, fill up the resulting opening in the walls of the stomach. The destruction sometimes extends to the organ which covers the ulcer, but more frequently a thick layer of connective tissue develops on the surface of this organ, and forms the floor of the ulcer. The covering organ never lies in the same plane with the inner wall or projects into the stomach. But, after the muscular coat has retracted, the mucous coat becomes everted at the edge of the ulcer, and comes in contact with the organ in question. If, in such cases, the ulcer heal, the connective-tissue layer on the organ contracts, the edges approach each other, and, if the opening be not too large, may finally unite so as to form a firm, hard cicatrix.

When the ulcer first forms, and still more frequently while an ulcer already formed is spreading, the vessels of the stomach, or of the neighboring organ into which the ulcer has perforated, are destroyed, and there is considerable hæmorrhage into the stomach. Perforations of the coronary, pyloric, gastro-epiploic sinistra, gastro-duodenal arteries and their branches, of the splenic artery, but most frequently of its branches going to the pancreas, and of the pancreatico-duodenalis, have been observed.

The gastric mucous membrane also exhibits the changes characteristic of chronic gastric catarrh, which were described above. Sometimes these are absent or very slight.

Symptoms and Course.—Sometimes, by perforating all the coats, and thus permitting the escape of the contents of the stomach into the peritonæum, ulcer of the stomach may cause fatal peritonitis; or, by erosion of a large vessel, may cause abundant hæmatemesis before the disease has been recognized, or before its recognition was possible. It is going too far, however, to say that in such cases the signs of the suddenly occurring peritonitis, or the hæmatemesis, were the first symptoms of the ulcer of the stomach. On more careful inquiry, we almost always find that slight disturbances of digestion, and some oppression in the epigastrium, increased by eating, have gone before, and that the patient had been troubled by wearing any thing tight about the waist. Between the first appearances of these insignificant difficulties and the fatal termination, there is sometimes only an interval of a few days or weeks, so there can be no doubt that, in this short time, all the coats of the stomach have been perforated. (I have had a very sorrowful opportunity of satisfying myself of the rapid course of a perforating ulcer; in Magdeburg, Dr. *Brunnemann*, a very distinguished and promising young physician, died of such an ulcer. When the perforation occurred, he was not for an instant in doubt about the diagnosis, and most decidedly said that he had not suffered over eight days from slight trouble, which he thought proceeded from a slight gastric catarrh.) It even seems as if perforation, with escape of the contents of the stomach into the abdomen, occurred most frequently in the cases beginning in this concealed manner, and running a rapid course; that, on the other hand, in the cases which begin with severe and pathognomonic symptoms, and run on for months or years, the stomach has time, as it were, to unite to the neighboring organs, and so prevent the escape of its contents into the abdomen. I would remind my readers that the cheesy infiltrations of the lungs, which run a rapid course, lead to perforation of the pleura and pneumothorax far more frequently than miliary tuberculosis, which has a slow course, and where the folds of the pleura almost always become adherent, if the destruction goes as far as the pleura. The cases where the inconvenience is so slight that a certain diagnosis of ulcer of the stomach is impossible, or where the patient is so little troubled that he does not seek medical aid before the occurrence of the perforation, or the hæmatemesis, are, however, rare when compared to those where the disease is readily recognized, and where it excites very annoying symptoms. Among the most frequent and troublesome symptoms of chronic ulcer of the stomach are pains in the epigastrium. The patients complain partly of a steady pain in the pit of the stomach, which is increased by pressure, and is generally particularly severe at some circumscribed spot; partly of paroxysms of severe pain, which, starting from the epigastrium, extend toward the back, and are designated as attacks of cardialgia. The sensitiveness to pressure in the epigastrium, when the ulcer is extending in breadth or depth, is sometimes so great that the patient can hardly bear even the pressure of light bed-clothes; this is because there is slight peritonitis over the affected part. The cardialgic attacks generally occur soon after meal-times, and are severe in proportion to the coarseness and roughness of the food that has been taken. The patients sigh, groan, double themselves up, and often do not find ease till the stomach has been emptied by vomiting; if there be no emesis, the attacks of pain may last for hours. The seat of the ulcer may be determined with some certainty from the length of time at which the pains follow the meal; if they come immediately after eating, we may suppose that the ulcer is near the cardiac orifice; if they come an hour or two later, it will probably be in the pyloric portion. Although, as a general rule, the attacks of pain occur after eating, and are the more severe the more indigestible and the rougher the food, there are some exceptions, and it is important that we should know these even if we cannot explain them. In these exceptional cases, while the stomach is empty there is pain, which is relieved by taking food; or after eating indigestible food the patient remains free from pain, while it becomes very severe if he eat easily-digested articles. The attacks of pain are usually attributed to the irritation of the surface of the ulcer by the motion of the contents of the stomach; while in an empty stomach such causes are absent. Another explanation is that the gastric juice secreted on the introduction of food irritates the ulcer and excites the pain, while there are intermissions, because, while the stomach is empty, a mucus which is but slightly irritating covers the ulcer. But when we consider that perforation of all the coats of the stomach may occur without exciting these attacks of pain, and that, on the other hand, the most severe pain often continues when the ulcer has healed, but the stomach has become adherent to other organs, there seems no doubt that the chief if not the only cause of pain is the obstruction to the peristaltic movements of the stomach, due to cicatricial contraction, or the adhesion of its wall to neighboring organs. The larger and rougher the ingesta, the more energetic and continued are the movements of the stomach they excite; hence the severity and long duration of the paroxysms of pain after eating large pieces of bread, potatoes, and other vegetables, and the comparative ease of the patient after eating soup, milk, and other fluid and mild nutriment.

*Vomiting* is almost as constant a symptom as the sensitiveness of the epigastrium and the cardialgic attacks. It is caused by the same circumstances as the attacks of pain, and often terminates these, as it were. Vomiting also occurs a longer or shorter time after meals, according as the ulcer is near the cardiac or pyloric orifice. It is the more apt to occur, the nearer the ulcer is to the orifice of the stomach. *Henoch* calls attention to the fact that the same holds good in other hollow organs; that is, reflex movements are particularly liable to be excited in them by affections near their openings; he reminds us that severe spasm of the bladder is most apt to occur from inflammatory irritation about its neck; that tenesmus, depending on affections of the rectum, is more distressing the nearer the disease is to the anus. Patients usually vomit their food more or less changed, and mixed with mucus and sour fluids. The state of the substances vomited, in which there are often sarcina, depends principally on the intensity and extent of the coexistent gastric catarrh. Sometimes only quantities of mucus and acid fluids are vomited, while the food remains in the stomach.

Severe cardialgia and vomiting, occurring regularly after meals, render it very probable that there is a chronic ulcer of the stomach; the diagnosis is rendered certain, if there be also vomiting of blood. The hæmatemesis may have various sources: sometimes it is due to capillary hæmorrhage, induced by the spreading of the ulcer; more frequently it is caused by the erosion of a large vessel, and this form is pathognomonic of ulcer of the stomach. We shall speak more in detail of hæmorrhage from the stomach in Chapter VIII.

The symptoms of the chronic gastric catarrh which accompanies ulcer of the stomach unite with the characteristic symptoms of the latter, it is true, but they are more or less decided according to the grade and extent of the catarrh, sometimes being just apparent. Some patients have decided swelling in the epigastrium, frequent eructation, severe heartburn, complete loss of appetite; others feel very well during the intervals of their pain; even their appetite is scarcely impaired.

The signs of oral catarrh, which also complicates ulcers of the stomach, are somewhat different from the usual state of the mouth in chronic oral catarrh. The acid fluids that rise into the mouth appear to dissolve the epithelium and the vomiting to clear it away; at least, instead of the thickly-coated tongue, which is rarely absent in simple chronic catarrh of the stomach, we usually find the tongue red and furrowed, and this state is almost always accompanied by increased thirst. Finally, as in most cases of chronic gastric catarrh, there is habitual constipation.

Regarding the *general health* of the patient, chronic ulcer of the stomach may soon impair the nutrition, so that the patient is rapidly debilitated, and has a pale, cachectic look; in other cases, the nutrition is very little deranged. This difference unmistakably depends on the varied grade and extent of the accompanying gastric catarrh.

Except in the first-mentioned cases, where the round ulcer proves fatal in a few days or weeks, the course of the disease is usually very tedious; it may run on for years. During this time the symptoms may vary greatly. At one time the patient lives comfortably; at another, without apparent cause, he suffers severely. Not unfrequently, in the midst of an apparent convalescence or complete cure, vomiting of blood suddenly occurs; or the affection returns with its former severity years after it had disappeared.

Recovery is the most frequent termination of chronic ulcer of the stomach. The sufferings of the patient gradually subside, the nutrition is fully restored, all disturbance ceases, and when the patient has died of some other disease, we find the characteristic cicatrix as the sole remains of the ulcer.

Secondly, the result in incomplete cure is not infrequent. The symptoms of chronic gastric catarrh disappear, it is true; often also the periodical vomiting; the patient may become fresh and healthy-looking; but every meal is followed by cardialgia, which occasionally becomes more severe than usual. In such cases the ulcer has healed, and the gastric mucous membrane has become relatively healthy; but there is a cicatrix or more frequently an adhesion of the stomach to some neighboring organ, which limits its movements at some point, and keeps up the cardialgic attacks.

In other cases, ulcer of the stomach causes death. This may occur (*a*), from perforation of the walls of the stomach, and escape of its contents into the abdominal cavity. In such cases patients sometimes die before the occurrence of peritonitis, or before this has developed sufficiently to be regarded as the cause of death. Along with the sudden occurrence of fearful pain in the abdomen, the skin becomes cool, the pulse small, the countenance sunken; and the patient collapses and dies in this state. If the heart's action becomes weaker, the filling of the arteries less complete, typical cyanosis may result from the collection of blood in the veins, and the patient looks like one in the collapsed stage of cholera. In these cases the perforation appears to cause a paralysis in the sympathetic nervous system, analogous to what occurs in other severe injuries. Although such cases are not rare, it is more common for the patient not to die during the first day or two, but for the symptoms of a fatal peritonitis to combine with the above. (*b.*) In rare instances death results from hæmorrhage from the stomach. Even when the patient appears quite bloodless and waxy-looking, where every attempt to raise the head induces fainting, where oppression, palpitation, dizziness, tinnitus, and other symptoms of loss of blood, are present, the patient often recovers, contrary to all expectation. Death may result very quickly, however, from erosion of large arteries. I saw one case where the splenic artery was perforated, and the patient suddenly fell and died before there was any vomiting of blood. (*c.*) Death may result from gradual exhaustion, and this may take place even where the ulcer has healed, but there is a stricture of the stomach from cicatricial contraction. In such cases not only is there the severest cardialgia, but every thing that the patient eats is vomited; he may have no passage from his bowels for weeks, the belly sinks in, he wastes away to a skeleton, and dies from inanition.

DIAGNOSIS.—In the rare cases of ulcer of the stomach, where it runs its course without any pathognomonic symptoms, it cannot be distinguished from chronic gastric catarrh; but in most cases the differential diagnosis between the two affections presents no difficulty. Great sensitiveness of the stomach at a circumscribed spot, severe cardialgic attacks, but particularly vomiting of blood, exclude simple catarrh with great certainty. A far less certain sign is the appearance of the tongue, which is red and smooth in cases of round ulcer, and almost always coated in cases of simple catarrh. It may be difficult to distinguish it from stricture of the pylorus due to hypertrophy of the membranes of the stomach. The slightness of the cardialgic attacks, which are not in proportion to the frequency of the vomiting, the regular occurrence of the latter, and the presence of consecutive dilatation of the stomach, aid somewhat in the diagnosis, as they indicate stricture rather than ulcer.

Where, with great severity of the cardialgic attacks, there are no dyspeptic symptoms, and, in spite of the long continuance of their disease, the patients preserve a blooming appearance, we may suspect a cicatrix, which impairs the motions of the stomach. The probability of this is still greater if there have formerly been for a long time sure signs of an ulcer of the stomach, which, except the cardialgia, have subsequently entirely disappeared. Where the symptoms of stricture have developed and slowly increased after there has been chronic ulcer of the stomach, we must suspect that a cicatricial stricture has formed.

We will speak in the following chapter of the diagnosis of chronic ulcer of the stomach, from cancer and nervous cardialgia.

PROGNOSIS.—In accordance with what we have said of the course and results, the prognosis of chronic ulcer of the stomach is, on the whole, favorable; we must not forget, however, that the disease often has remissions followed by exacerbations, that in the midst of apparent improvement hæmorrhages will occur, and that, even after recovery has begun, relapses are always imminent.

TREATMENT.—The disease of the blood-vessels, which, as we have said, gives rise to the partial necrosis of the walls of the stomach which results in round ulcer, can rarely be referred to chronic gastric catarrh; for topers, who have the most obstinate forms of this disease, rarely have ulcer of the stomach. As we do not know the causes of these affections of the blood-vessels, we have no hesitation in saying that, in the treatment of chronic ulcer of the stomach, we cannot fulfil the *causal indications.*

*Dietetic rules* best answer the indications from the disease. The result of the treatment mostly depends on their being strictly followed out. It is true, we cannot protect the affected portion of the wall of the stomach from injury, as we would an ulcer of the skin; the introduction of even the mildest food excites a hyperæmia of the gastric mucous membrane and irritates the affected part; however, the coarser and rougher the ingesta, the greater the irritation they excite. From this fact, proved by experiment and confirmed by practice, we deduce the rule that the patients should have the mildest possible, and, preferably, a liquid diet. We should then try if an exclusively milk diet will be borne; unfortunately, this is not always the case. If fresh milk curdles in the stomach to hard, tough lumps, we should always give it with white bread, as advised by *Budd.* Some patients, who cannot use fresh milk, have no difficulty with buttermilk, or sour milk. If the patient has a great distaste for milk-diet, or if he cannot take sour or buttermilk, we may give rich unskimmed soups, with an addition of *Liebig's* meat-extract. The nutrition in the small quantities of these extracts that the patient takes is not great, it is true, but they are strong analeptics. *Trommer's* malt-extract, which has been before described, contains the nutritious constituents of malt in a state of solution, and is to be recommended because several spoonfuls of it may be taken daily without difficulty; hence it must be regarded as a valuable remedy. I know patients who have taken one or two ounces of *Trommer's* malt-extract daily for years. Vegetables, bread from unbolted flour, roast potatoes, potato salad, etc., are particularly to be avoided, while purée of potatoes does well.

The therapeutic use of the alkaline carbonates has a wonderful effect in chronic ulcer of the stomach. Among the mineral waters containing alkaline carbonates and purgative salts, the warm springs are preferable to the cold. Patients whose circumstances allow it may be sent to Karlsbad. If there be insuperable obstacles to a trip to the springs, Karlsbad, Marienbad, Tarasper, and similar waters may be prescribed at home; they should be properly warmed before drinking. In some cases I have seen patients wonderfully improved by the treatment at Wildbad, and other Akrato-therma, after they had taken the waters of Karlsbad and Marienbad without benefit; but am not convinced that the use of water from warm springs can replace the treatment at Karlsbad, as Professor *Bock* claims in the "Gartenlaube." I am sorry that so clear and shrewd a person as *Bock* should permit himself to spread a dangerous half-knowledge among the laity; I have more than once found that people, under the impression that they had learned enough from *Bock's* writings to judge of their cases and treat themselves, have done themselves great injury. The rules of the water-cures contain some superfluous and finikin regulations, but it is not well to shake faith in these, or else even the rational rules may be less carefully followed. Among the latter I place in the first rank, that patients should not eat later than seven o'clock, and then only soups, that they should not breakfast for at least half an hour or an hour after their last glass of water. It is certain that very much depends on the warm mineral water going into an empty stomach. If under the treatment just described, contrary to our expectations, there be no improvement, we may give nitrate of silver, or subnitrate of bismuth. From the effect that these remedies, particularly the former, are seen to have on ulcers of the skin, or of other mucous membranes, their use appears perfectly rational, and in some cases the results attained with them are surprising. In other cases, on the contrary, they do no good. For the mode of administering these remedies and their dose, we refer to what was said in the treatment of chronic gastric catarrh.

The treatment of symptoms, first of all, requires the relief of the cardialgic attacks. There are but few cases of chronic ulcer of the stomach where we can dispense with the use of narcotics; these usually have an instantaneous and brilliant effect on the attacks of pain. Even a few minutes after the administration of a small dose of morphia, there is relief or even complete freedom from pain. This seems to show that the pain is chiefly induced by tension of the stomach. If it depended on irritation of the walls of the stomach by the ingesta, or the gastric juice, we could not explain the action of the narcotics, which *Jaksch* says is often magical; but, if it depends on tension of the walls of the stomach, we can readily understand the effect of narcotics, which, besides their anæsthetic effect, retard the movements of the stomach. *Stokes* declares that morphia is the only trustworthy remedy in the treatment of chronic ulcer of the stomach, and thinks that all the other remedies which are highly spoken of are only effectual when combined with a narcotic, as is generally done in using subnitrate of bismuth. Very small doses ($\frac{1}{18}$—$\frac{1}{8}$ gr.) of morphia usually suffice, and it is not necessary to increase these. *Jaksch* saw a woman take the same sized dose of morphia more than a hundred times without its efficacy diminishing. Morphia is preferable to extract of hyoscyamus or belladonna, which are also recommended. When the epigastrium is very sensitive to pressure, a few leeches or wet cups fulfil the indications. If these do not answer the purpose, blisters or pustulating plasters left for some time on the pit of the stomach may be of use. Among the symptoms deserving particular attention, we may have obstinate vomiting. The narcotics, particularly morphia, seem to be of aid in this symptom also. If they do no good, small pieces of ice or mouthfuls of ice-water are sometimes beneficial, and occasionally where all else fails we may give creasote (gtt. jv. to ℥ vj. of water, in tablespoonful doses), or tincture of iodine (gtt. ii.—iii. in sweetened water). Finally, in the course of chronic ulcer of the stomach, hæmatemesis or peritonitis may require special treatment; but we will speak of this in the chapters devoted to these subjects.

## CHAPTER VII.

### CARCINOMA OF THE STOMACH.

ETIOLOGY.—Among the internal organs, the stomach is the one most frequently affected with carcinoma; it is usually affected primarily; more rarely it is secondary to cancerous degeneration of other organs, or is propagated from them to the stomach.

The cause of cancer of the stomach is just as obscure as that of cancer elsewhere. In some families it seems to be hereditary: the father of Napoleon I., his sister, and himself, died of this disease. In regard to the influence of sex, age, and employment, we may say that men are more frequently affected than women; that it is most frequent between the ages of forty and sixty years, while before forty it is rare, and before thirty it very exceptionally occurs; finally, that no class of people escape it entirely. If cancer of the stomach be more frequent in the inferior classes than in the higher, it is because the former are more numerous. There is no proof of what has been said of the influence of strong liquors, mental depression, the suppression of eruptions, or the cure of ulcers (*J. Frank*).

ANATOMICAL APPEARANCES.—Cancer of the stomach most frequently attacks the pyloric portion of the stomach, less often the cardiac portion or the small curvature, and most rarely the fundus and greater curvature. It always shows a tendency to spread transversely, so that cancer of the small curvature extends toward the greater, and that of the pyloric or cardiac portions readily causes annular stricture. Cancerous stricture of the pyloric portion is usually sharply limited by the pylorus, while that of the cardiac almost always affects more or less of the œsophagus.

Of the various forms of carcinoma of the stomach, the most frequent is scirrhus, medullary next, and the least frequent are alveolar or colloid; the various forms often combine—the union of scirrhus and medullary is most common.

*Scirrhus* almost always begins in the submucous tissue; it occasionally forms small nodules, sometimes diffuse thickening, which, growing irregularly, gives an uneven appearance. The growth has the characteristics of hard cancer, and presents a dull, whitish, dense mass of cartilaginous hardness. The mucous membrane soon unites with the subjacent neoplasia; it subsequently softens to a black pulp, sloughs off, and the bare surface of the cancer is left. The muscular coat usually becomes hypertrophied, and shows the previously-described fan-like appearance; it may subsequently atrophy under the pressure of the growth, or may be destroyed by the latter. The serous coat becomes thickened and clouded by local peritonitis, often unites with adjacent parts, and is frequently covered with milky, laminated deposits. After the destruction of the mucous membrane, the exposed cancer begins to ulcerate; at first shallow, later deep excavations are formed, and we have an irregular cancerous ulcer, with hard, callous edges, like those occurring on the skin. In other cases medullary masses spring from the floor and borders of the scirrhous ulcer.

If the affection of the stomach commence as *medullary* cancer, the nodules and diffuse thickening of the submucous tissue are softer from the first; they look like brain-substance, and, after a section through them, we may press out a quantity of the so-called "cancer-juice." Medullary cancer spreads much more rapidly than scirrhous, and soon projects from the inner surface of the stomach as soft, easily-bleeding, spongy excrescences. The middle of the growth usually breaks down into black, soft, ragged masses, while the proliferation continues at the periphery. If the dead masses be thrown off, there is left an excavated ulcer, surrounded by elevated, everted, cauliflower edges.

Such a cancerous ulcer may attain twice the size of the hand, and the proliferations be sufficient to encroach considerably upon the space in the stomach.

*Alveolar* or *colloid* cancer rarely appears as scattered nodules; it more frequently occurs as diffuse degeneration. It also usually commences in the submucous tissue, but soon induces degeneration of all the coats of the stomach; in the wall, which has become several lines, or even half an inch thick, no trace of the original structure can be found; it consists almost entirely of innumerable small cavities (alveoli), which contain a gelatinous fluid. Microscopic examination of the latter shows the cellular formation characteristic of gelatinous cancer. In alveolar cancer, also, the mucous membrane is destroyed, the alveoli are evacuated, the free surface appears ragged and discolored; but the loss of substance never becomes very deep, for, while the destruction goes on above, there is new production below.

The cancerous degeneration often extends to other organs, particularly to the lymphatic glands, the pancreas, liver, transverse colon, or omentum. The breaking down of the growth may also extend beyond the stomach to these organs, and give rise to communication between the stomach and the intestines, or, after adhesion of the stomach to the anterior wall of the abdomen, perforation outwardly may occur. Alveolar cancer alone rarely affects the organs above mentioned; but it frequently induces diffuse degeneration of the peritonæum and consequent ascites.

If the breaking down of the cancer advances to the peritonæum before the stomach has become adherent to the neighboring parts, its contents may escape into the abdomen, and fatal peritonitis result.

If the cancerous degeneration leads to stricture of the pylorus, which, by nodular prominences and angular curvature of the contracted portion may still more increase the difficulty of the escape of the contents of the stomach, dilatation of the stomach will result. If, on the other hand, the cancer be located at the cardiac portion, or, if there be degeneration of a large portion of the wall of the stomach, as happens particularly in alveolar cancer, the stomach may become smaller.

In many cases the diseased pylorus remains at its normal locality, being attached by adhesions; but in many others it moves freely, and, from its weight, sinks down in the abdomen even as low as the symphysis pubis.

SYMPTOMS AND COURSE.—Cases occur where it is impossible to recognize cancer of the stomach with certainty during life. In eleemosynary and hospital practice, patients not unfrequently come under treatment who are emaciated to skeletons, indifferent, and so apathetic, that they make no complaints, and cannot give any history of their case. At the same time they have no pain on pressure in the epigastrium; they have little appetite, but the food taken is not vomited, and examination of the abdomen reveals no tumor. It is necessary to know that cancer of the stomach may occur without the last-mentioned symptoms, and remember the possibility of its being the cause of the excessive marasmus; but an absolute diagnosis is impossible. If such a patient die and at the autopsy a large cancerous ulcer of the stomach be found, inexperienced physicians are usually greatly astonished, and cannot understand how a disease so severe and so far advanced could be mistaken.

In other cases we can make an approximative diagnosis of cancer of the stomach. A patient, far advanced in life, complains of loss of appetite, of a feeling of pressure and fulness in the epigastrium, of eructation and other dyspeptic symptoms; but, along with these mild symptoms, the patient rapidly loses his strength, acquires a dirty-yellow cachectic color of the face, and finally œdema of the ankles occurs. If, in such a case, we can exclude other diseases, which might explain the cachexia and marasmus, we have cause to suspect that there is not a chronic catarrh, but a malignant disease of the stomach, even if there be no actual pain, vomiting, or characteristic tumor.

In most cases the symptoms of cancer of the stomach are much more decided, and can scarcely be mistaken. Besides the dyspepsia and symptoms of cachexia and marasmus, there is *tenderness* in the region of the stomach. This is increased by pressure, and also after eating; but does not usually attain the severity of cardialgic attacks. Almost as often there is *vomiting*. When the cancer is in the smaller curvature, the vomiting only occurs now and then, but comes after every meal, when it constricts the pyloric or cardiac orifices. In stricture of the pylorus the vomiting does not usually come on till several hours after eating; in stricture of the cardiac orifice it occurs immediately after or during eating. Occasionally it happens that, after the vomiting has recurred regularly for a time, it gradually becomes less frequent, ceases altogether, or is replaced by a sort of chewing the cud. This occurrence is explained when the autopsy shows that by breaking down of the cancer the contracted part has become larger, or that the stomach, being enormously dilated or structurally changed, is evidently not in condition to contract and perform its part in the act of vomiting. In other cases the autopsy gives no explanation of the cessation of vomiting. The vomited masses consist sometimes of the food, enveloped in a thick coat of mucus, which is little changed, if the cancer be at the cardiac extremity, but is often greatly altered when the pylorus is affected; sometimes they consist only of quantities of mucus and variously-colored sour and bitter liquid. The presence of lactic, butyric, or acetic acids in the vomited matters depends on the causes that we investigated when speaking of simple stricture of the pylorus. Sarcina ventriculi very often occur in the vomited matters, while portions of the cancerous growth are rarely found. This is because, while the cancer ulcerates and breaks down, the characteristic form of its elements is destroyed. Capillary hæmorrhage readily results from the breaking down of the vascular growth. The blood poured into the stomach is quickly altered by its acid contents and converted into a black, grumous mass. Hence the presence of "coffee-ground" masses in the vomited matters is a frequent and important symptom in cancer of the stomach; but its diagnostic significance has been much overestimated. The erosion of large vessels, causing copious hæmorrhage, is much more rare; in doubtful cases this symptom indicates chronic ulcer of the stomach (see Chapter VIII.) rather than cancerous disease.

The most important symptom of cancer of the stomach is the presence of a tumor in the epigastrium. It is necessary to know that this symptom may be wholly absent; this may be readily understood when we remember the relation of the stomach to the liver and ribs. Cancer of the cardiac portion never causes a perceptible tumor, even when it attains a great size; that of the small curvature is not felt till it has advanced to the greater curvature. Most of the tumors that can be felt are located at or near the pylorus, the pyloric part of the stomach; and it is only because cancer affects this portion most frequently, that we almost always find a tumor in this disease of the stomach. *Hyrtl's* description of the position of the stomach is not true; this is particularly the case in the assertion that, on expiration, the point of the sternum corresponds to the middle of the anterior wall of the stomach. *Luschka* says, in his classical work (on every page of which the practitioner will find valuable information), that an incision made through the median line of the body will divide the stomach, so that five-sixths will lie on the left side, and at most one-sixth on the right. When the stomach is in a normal position, even cancer of the pylorus will give a tumor of the left side. When the diseased pylorus sinks down from its weight, the tumor may be near the navel, usually somewhat above and rather to the right than to the left of this. If it be still farther down, it may be mistaken for ovarian tumor. The size of the tumor varies from that of a pigeon's egg to that of the fist; if it be very large, it may form a visible prominence in the abdomen; the surface is usually uneven and nodular. In many cases the tumor is movable, and it changes position according as the stomach is full or empty; in other cases, where there are adhesions, it is immovable. In the same way the sensitiveness of the tumor varies greatly. Sometimes, instead of a circumscribed, nodular tumor, we find a regular, more or less extensive prominence and resistance of the epigastrium. Percussion of the tumor, caused by cancer of the stomach, almost always gives a not quite clear and decidedly tympanitic sound.

In cancerous stricture of the pylorus, besides showing the presence of a tumor, physical examination may prove a dilatation of the stomach, whose symptoms we described while speaking of simple stricture of the pylorus; if, on the other hand, the stomach be contracted, and, as often happens, the bowels be empty, the lower margin of the ribs becomes very prominent, while the belly is sunken, so that we can distinctly feel the vertebral column and the pulsating aorta. The symptoms of cancer of the stomach are modified by the development of cancer in other organs, particularly in the liver. Occasionally, also, cancer of the lymphatic glands of the stomach spreads to the retroperitoneal glands, to those of the mediastinum, thence to those of the neck, so that a hard swelling of the supra-clavicular glands may be of diagnostic importance in cases of cancer of the stomach (I have seen such cases). The symptoms of alveolar cancer are often modified by the occurrence of ascites.

During the course of cancer of the stomach the symptoms generally increase regularly; more rarely, the patient improves for a time, pain and vomiting cease for a while, and even the appetite returns. These remissions do not usually continue long; the difficulties increase again, the appetite is entirely lost; constipation, which has existed from the first, can hardly be overcome; emaciation makes giant strides. If the cancer be medullary, the disease usually runs its course in a few months, while scirrhus, and particularly alveolar cancer, may run on for years.

The only termination of cancer of the stomach is in death. In those cases where cure of cancer has been claimed from the clinical course of a disease of the stomach, there may have been a mistake in diagnosis. Those observations, where autopsy is said to have revealed the cicatrix of a cancerous ulcer, also are not perfectly trustworthy; for if fresh cancerous growths are found in the vicinity of the cicatrix, the disease has not truly disappeared; but if this support be wanting, the cicatrix from a cancer cannot be certainly distinguished from that of a simple ulcer. In most cases, death occurs with the symptoms of exhaustion. As there is no accompanying fever, the last stage of the disease is usually much protracted, and patients may live for days, while we hourly expect their death. In these cases the tongue usually becomes red, inclined to dryness, and covered with aphthous deposits. Besides this painful affection, there is not unfrequently a painful tense œdema of one of the legs shortly before death. This symptom depends on obstruction of the femoral vein, and shows that, in consequence of the retarded circulation, a clot has formed in it. More rarely death occurs from a rapid peritonitis, after perforation of the stomach. Still more rarely, copious hæmorrhage from the stomach hastens the exhaustion, or, by its extent, causes sudden death. Finally, death may be caused or hastened by complications and secondary diseases.

DIAGNOSIS.—In cases where the epigastrium is painful, where there is frequent vomiting, occasionally of substance looking like coffee-grounds, where there is a tumor in the epigastrium, the diagnosis of cancer of the stomach from *chronic catarrh* is easy. If these symptoms be absent, particularly the tumor, which *Andral* maintains is the only certain sign of cancer of the stomach, the differential diagnosis of these two diseases may be very difficult. In forming an opinion, the age of the patient is important; besides this, we often can only judge from the general condition.

The diagnosis from chronic ulcer of the stomach, also, is sometimes easy, again very difficult. In one well-known case, two medical celebrities, *Oppolzer* and *Schoenlein*, could not agree whether there was cancer or ulcer of the stomach. In the differential diagnosis, the factors chiefly to be regarded are the following: 1. The age of the patient; in young persons, cancer may be excluded almost with certainty. 2. The duration of the disease; if it has existed more than a year, the probabilities are against cancer. 3. The strength and condition of the patient; in ulcer, these are often affected but little, and not till late in the disease, while in cancer they are greatly impaired very early. 4. The character of the pain; cardialgic attacks indicate ulcer rather than cancer. 5. The condition of the blood vomited; in ulcer of the stomach, it is in large quantities, and hence slightly changed, while in cancer the amount is usually scanty, it is black, and looks like coffee-grounds; still, in some cases of cancer, there is abundant vomiting of blood, and with an ulcer of the stomach there may be ejection of black, grumous masses. 6. The presence or absence of a tumor; the former proves almost certainly that there is cancer, for the cases where thickening of the walls of the stomach and connective-tissue growths, in the vicinity of an ulcer, cause a tumor, are exceedingly rare. But, on the other hand, we must not forget that absence of a tumor does not prove that there is no cancer.

The variety of the cancer can rarely be determined with any certainty during life. Alveolar cancer, being the rarest variety, is only to be suspected where the disease runs a very slow course, and when there is ascites. The diagnosis becomes more certain if, after tapping, nodular masses can be felt in the omentum. If these symptoms are absent, we must suspect scirrhus or medullary cancer. The more acute the course, the larger the tumor becomes and the more rapidly it grows, the more probable it is that the new formation is medullary cancer.

TREATMENT.—As the indications from the cause and from the disease cannot be fulfilled, we must restrict ourselves to the treatment of symptoms. The diet must be regulated according to the rules prescribed in the treatment of chronic gastric catarrh; if milk be well borne, it is the most suitable nourishment; if milk cannot be used, concentrated broths, yolk of egg, and other nutritious substances may be ordered, but always in small quantities, and where there is stricture they should be given in fluid form or finely divided. Wine also, particularly red wine, may be given, and it is usually well borne. For the excessive acidity, the alkaline carbonates do well, especially in the form of soda-water; they often fail, however, and sometimes, as in stricture of the pylorus, we cannot in any way prevent the acidity. In such cases it is well at each meal to give pills containing gtt. 1—1½ of creasote, as recommended by *Budd*. Pills of aloes and colocynth may be given for the obstinate constipation. For the severe pain and the sleeplessness, narcotics, particularly morphia, must be given.

## CHAPTER VIII.

### HÆMORRHAGE FROM THE STOMACH.

ETIOLOGY.—1. Hæmorrhage from the stomach results from rupture of the over-filled blood-vessels without previous change of texture. *Arterial fluxion* is rarely so decided as to cause rupture; besides the slight hæmorrhage seen in inflammation of the stomach, this occasionally occurs in anomalies of menstruation. It cannot be denied that ripening and detachment of an ovum are sometimes accompanied by fluxions and hæmorrhages in other organs and not in the uterus, although we can give no explanation of the fact. *Venous congestion* of the gastric mucous membrane is a much more frequent cause of hæmorrhage. The most decided congestions result from impediments to the circulation in the liver. Hæmorrhage from the stomach may be caused by obstructions of the portal vein by blood-clots; by pressure on its branches due to cirrhosis of the liver, or to the enlargement of the gall-ducts, caused by closure of the ductus hepaticus or choledochus; by plugging of the capillary vessels of the liver with clumps of pigment in pernicious fever (*Frerichs*); finally, by destruction of the capillaries, in the so-called yellow atrophy of the liver. The hyperæmia of the gastric mucous membrane, due to obstruction of the circulation in the chest by diseases of the lungs, pleura, heart, or pericardium, is rarely sufficient to cause a rupture of the vessels; but hæmorrhage from the stomach is sometimes seen from these causes. The hæmorrhages which sometimes occur in new-born infants most likely belong in this class. It is most probable that these depend on imperfect expansion of the lungs, and the obstruction thus induced to the flow of blood from the stomach.

2. Hæmorrhage from the stomach may result from the rupture of diseased vessels. In rare cases varices burst or aneurisms open into the stomach. More frequently disease of the walls of the vessels must be suspected, without our being able to prove it either with or without the microscope. Under this head come the hæmorrhages occurring in the so-called hæmorrhagic diathesis; those coming after exhausting diseases, in the course of yellow fever and other severe diseases; finally, those arising from improper living, especially abstinence from fresh meat and vegetables, which form one of the symptoms of scorbutis. In these cases it is insufficient to ascribe the bleeding immediately to an abnormal quality of the blood; this can only act by disturbing the nutrition of the walls of the vessels.

3. Finally, hæmorrhage from the stomach may arise from erosion and other injuries of the walls of the vessels. In this class belong the cases where chronic ulcer or ulcerating carcinoma leads to hæmorrhage from the capillaries or larger vessels; those where corrosive substances and sharp, foreign bodies open vessels of the stomach; lastly, those where a blow over the stomach has caused rupture of one of the vessels.

Anatomical Appearances.—Even after decided hæmorrhages from the gastric mucous membrane, we often seek in vain, on *post-mortem* examination, for its source; when the patient has died from loss of blood, after washing off the stomach, we find it just as pale and bloodless as the rest of the body. In other cases there has been coincident capillary hæmorrhage in the mucous membrane, in which we find bluish-red or blackish-red spots, from which blood oozes out on slight pressure. This hæmorrhagic infiltration of circumscribed portions of mucous membrane generally leads to superficial softening and throwing off of the softened portion; superficial excavations thus occur, which are not discovered till the dirty-brown blood particles clinging to them are washed off. The superficial bleeding fossæ, called hæmorrhagic erosions, are usually numerous, small, of round or elongated form, and are chiefly found at the summit of the longitudinal folds formed by the mucous membrane. If large vessels have been eroded by ulcer or cancer of the stomach, or if ruptured varices or aneurisms have caused the hæmorrhage, we may in many cases find the gaping mouth of the vessel.

When the patient dies soon after the hæmorrhage, and if this has been very copious and occurred rapidly, the blood contained in the stomach forms red, clotted masses. If it has escaped slowly, and has been retained in the stomach a long while, so that the gastric juice and the acid contents of the stomach have had a chance to act on it, it appears brown or black. Where the hæmorrhage has been very slight, we find only a few black striæ and flocculi, or masses like coffee-grounds, in the stomach.

SYMPTOMS AND COURSE.—If the hæmorrhage from the stomach be not abundant and the blood be not vomited, the hæmorrhage is not usually recognized during life. According to *Beaumont's* observations, small hæmorrhages usually occur in the stomach during acute gastric catarrh; but blood mixed with mucus is very rarely vomited. The hæmorrhagic erosions also, which, as proved by autopsies, quite frequently accompany chronic catarrh, cancer, and ulcer, rarely cause hæmatemesis and consequently are rarely recognized during life.

In other cases, it is true, the mixture of small quantities of blood with the vomited matters leaves no doubt that there has been a hæmorrhage from the stomach, when it is certain that the blood has not been previously swallowed; but frequently hæmatemesis is the only symptom of the bleeding. This is daily observed in patients with cancer of the stomach, who are neither better nor worse when they vomit the "coffee-ground" masses.

If there has been a quantity of blood poured into the stomach, there are usually some symptoms preceding the hæmatemesis. These depend partly on the stomach being full of blood, partly on the emptiness of the blood-vessels of the body. The patients have a feeling of pressure about the stomach, a desire to loosen the clothes, feel constricted and nauseated; they become pale, the pulse is small, the skin cool, they see sparks before the eyes, have noises in the ears, become dizzy, or they even faint. I knew one case where a surgeon opened a vein for his mother, while she was in such a state, thinking that she was apoplectic. In robust, strong persons the faint feelings do not occur, and the premonitory symptoms are limited to the feeling of pressure and fulness in the epigastrium. After there has been nausea for a time, accompanied by the feeling of a warm fluid rising in the œsophagus, and a sweetish, stale taste, there is violent vomiting, and, to the great terror of the patient, blood, partly fluid, partly clotted, and usually dark brown, is evacuated through the mouth and nose. Small portions of blood often enter the larynx and induce coughing, and, as blood is brought up by this also, the patients relate that they have "broken a blood-vessel," but they cannot say whether they vomited or coughed up the blood. Sooner or later, after the hæmatemesis, there is a passage of blood from the bowels. If the hæmorrhage were very copious, blood is passed at stool very soon afterward, and it appears in black, clotted masses; if it be not passed for two or three days after the hæmatemesis, it is usually changed to a black, tar-like mass. In exceptional cases the blood poured into the stomach is evacuated by stool alone, and there is no vomiting. If patients, suffering from chronic ulcer of the stomach, become very pale in a short time, and show other symptoms that may depend on internal hæmorrhage, we should not neglect to examine the passages repeatedly. Many pounds of blood may be withdrawn from the circulation in a short time by hæmorrhage of the stomach; and even the strongest persons will then become pale, cool, and faint. In severe cases, every attempt of the patient to sit up, or even to raise the head, causes nausea, blackness before the eyes, and dizziness; every attempt to rise brings on fainting. Terrifying as it usually is to the patient and those around him, the fainting undoubtedly has a beneficial effect on the attack, for it momentarily arrests the hæmorrhage and favors the formation of coagula. It appears entirely due to this fact that the affection usually terminates more favorably than we should expect from the appearance of the patient. Indeed, proportionately few patients die of hæmorrhage from the stomach, that is, by bleeding to death, or suffocating from the blood entering the larynx. Much more frequently, after the patients have become deathly pale and excessively exhausted, and have lain for days in an apparently hopeless state, the vomiting ceases, blood gradually disappears from the stools, and a very slow convalescence begins. The patients long remain without appetite, complain of foul eructations and an unpleasant taste. As the great loss of blood is replaced by water, the patients become very hydræmic, and often dropsical; but these symptoms may also disappear, although, perhaps, somewhat slowly, and the patients recover.

Finally, we must mention those cases where the hæmorrhage is so profuse that the patient dies before the blood is evacuated either upward or downward. We must remember this when a patient, who has had the symptoms of chronic ulcer or cancer of the stomach, suddenly sinks with the symptoms of internal hæmorrhage and dies in a few minutes.

DIAGNOSIS.—Since patients with hæmoptysis often vomit at the same time, and those with hæmatemesis frequently have a coincident cough, it is not always easy to distinguish hæmorrhage of the stomach from that coming from the lungs or bronchi, particularly if we are not present at the time, or if it is a question of a "hæmorrhage" that has occurred some years before. The following points are important in the differential diagnosis.

1. The appearance of the blood ejected. Vomited blood is usually dark, blackish, clotted, mixed with food; the coagula, containing no air, are heavier; sometimes it has an acid reaction from the gastric juice. On the other hand, blood coming from the lungs and bronchi is usually bright-red, frothy, mixed with mucus, not coagulated at first, and if a coagulum does form, it contains air-bubbles and is light; its reaction is always alkaline. But we must know that blood, which has been but a short time in the stomach, and has been little affected by the gastric juice, may be bright red, and, on subsequent hæmatemesis, small portions of black blood may be thrown off.

2. Vomiting of blood is, in most cases, preceded by cardialgic attacks and other symptoms of ulcer or cancer of the stomach; in the much rarer cases caused by congestions and fluxions, there are symptoms of hyperæmia of all the organs in the abdomen; hæmoptysis, on the other hand, is usually preceded by disturbance of the respiration, and of the circulation in the thoracic viscera.

3. Intelligent patients can generally tell whether vomiting occurred first and was followed by coughing, or whether, on the other hand, nausea, retching, and vomiting have been excited by the coughing.

4. In hæmatemesis percussion generally shows fulness of the stomach, while physical examination of the chest shows no changes there. In hæmoptysis there is no epigastric dulness on percussion, and in the thorax we almost always hear moist *râles*, if there be no other sounds present.

5. After vomiting of blood, there are almost always bloody stools for a few days; after coughing of blood, instead of these, there is just as often a bloody mucous expectoration.

We cannot tell, from the vomited matters or from the stools, whether the blood vomited really comes from the stomach or whether it has been swallowed. In doubtful cases we should carefully examine the nose and pharynx, and ask the patient if he noticed any signs of nose-bleed before going to bed the previous night. A careful inquiry about premonitory symptoms may clear up the matter here also, particularly when we suspect intentional deception. In the description of the symptoms preceding the vomiting, malingerers usually overdraw the picture, and this fact, with the contradictory statements they make, often assists to expose them.

It is generally easy to decide whether the vomited matters be really blood or not, although there are cases where the physician has lost his presence of mind, and mistaken stewed cherries for blood. Even in the black coffee-ground masses some shrunken and misshapen blood-corpuscles can almost always be recognized with the microscope, and a chemical examination to prove the presence of iron in the black masses, will very rarely be necessary to determine that it is really altered blood.

It is usually easy to decide whether the bleeding depends on the erosion of large vessels or the rupture of capillaries. Besides the fact that in the first case the bleeding is usually more abundant than in the latter, a review of the premonitory symptoms almost always gives a certain means of diagnosis. If there have been cardialgic attacks, chronic vomiting, and other symptoms of ulcer of the stomach, there is probably erosion of a large vessel, which is by far the most frequent cause of hæmorrhage from the stomach. If, on the contrary, there have been ascites, enlargement of the spleen, or other signs of obstruction to the portal circulation, the hæmorrhage is most probably from the smaller vessels, and was caused by venous congestion. If the hemorrhages occur regularly every four weeks while there is amenorrhœa, we must suspect extensive fluxion to the stomach; if it come during yellow fever, or scorbutis, or after exhausting diseases, we must suspect disturbance of nutrition of the walls of the vessels.

PROGNOSIS.—We have already mentioned that only a small proportion of patients die from hematemesis, and that in spite of the waxy color of the skin, and even of the long-continued faintness, we may give a favorable prognosis. It is doubtful whether hæmorrhage from the stomach has under any circumstances a beneficial influence on chronic ulcer of the stomach; if the patients are occasionally better for a long time after it, it is probably because the severe attack has frightened them, and they have become more careful in their diet. The hæmorrhages caused by congestion may temporarily have a good effect on the other symptoms of abdominal plethora. On the other hand, in scorbutis and other exhausting diseases, hæmorrhage from the stomach always renders the prognosis more grave.

TREATMENT.—The prophylactic and causal indications are fulfilled by the treatment of the original disease. If patients, with cirrhosis or other disturbance of the circulation of the liver, show premonitory symptoms of hæmorrhage from the stomach, we may with advantage apply a few leeches to the anus; in women, who with amenorrhœa have periodical vomiting of blood, we may from time to time apply a few leeches to the os uteri.

The indications from the disease require a less energetic treatment in hæmorrhage from the capillaries, than in that depending on erosion of a large vessel. At the commencement of the latter, *Jaksch* recommends a venesection; but this is rarely beneficial, and when not so, increases the danger. The employment of *Junod's* cupping-boot would be much more advisable, but never after faintness has come on; for, after this, the use of hæmospastics, which may cause even robust patients to faint, is very dangerous. Cold must be regarded as the most efficacious remedy in hæmorrhage from the stomach; we may let the patients swallow small quantities of ice-water or small pieces of ice, from time to time, and we may cover the epigastrium with cold water or ice-compresses, and renew them frequently. Styptic medicines are not always well borne, but are often vomited up; the best of these are mistura sulphurica acida or alum, particularly in the form of serum lactis aluminatum. We should always give these remedies in small quantities and keep them on ice. Acetate of lead, sulphate of iron, and ergotin, may be dispensed with.

The indications from the symptoms, first of all, require attention to the syncope. The patient must lie flat in bed; must not rise to stool, but use a bed-pan. If syncope occur, we may hold eau de cologne or hartshorn to the nose, and sprinkle the face with water, but be very careful about the internal administration of restoratives. Among these cold champagne is best, as it is less apt to cause vomiting than the analeptic medicines. The unceasing inclination to vomit, which is partly due to the attacks of syncope, partly to the blood in the stomach, is the most annoying symptom that the patient has. In trying to arrest it, we should be careful about the use of narcotics, and should preferably apply a sinapism to the pit of the stomach occasionally, and give a pinch of effervescing powder. Since *P. Frank* has announced that it is necessary to purge patients with hæmorrhage from the stomach, to prevent the blood from exciting low and putrid fever, clysters and slight cooling laxatives are almost universally prescribed. My observations correspond with those of *Bamberger*, according to whom even enemata are injurious for the first few days after a hæmorrhage.

## CHAPTER IX.

### SPASM OF THE STOMACH—NERVOUS CARDIALGIA.

ETIOLOGY.—By nervous cardialgia, we mean painful affections of the stomach, not dependent on perceptible changes of structure. *Romberg* distinguishes two forms of this disease, one of which, he says, depends on a hyperæsthesia of the pneumogastric, the other on hyperæsthesia of the solar plexus. The former he calls gastrodynia neuralgica, the latter neuralgia cœliaca. But it can probably never be determined in any given case whether the pains be located in the filaments of the pneumogastric or in those of the sympathetic; and *Henoch* says, with truth, that this distinction is practically worthless, though it may be theoretically correct.

1. Like other nervous diseases, this affection is often observed in anæmic persons. If in chlorotic females, who have more or less severe attacks of spasm of the stomach as a constant symptom, the blood be enriched by preparations of iron, the cardialgia disappears, even in those cases where the amenorrhœa continues, and the quick recurrence of the chlorosis proves that the original disease has not been removed. It follows, from these observations, that the cardialgia of chlorotic patients depends solely on the poverty of the blood; not, as in hysterical women, on affections of the sexual organs. The cardialgia not unfrequently observed in tuberculous persons, convalescents, and onanists, probably, also, depends on poverty of the blood.

2. Diseases of the uterus, such as dislocations, flexions, or chronic inflammation, and follicular ulcers of the os uteri, as well as affections of the ovaries, induce cardialgia. It is among the most frequent symptoms of hysteria. The dependence of spasm of the stomach on affections of the female sexual organs is most evident when the attacks occur exclusively, or are most severe, at the menstrual periods. I treated one woman for amenorrhœa with retroflexion of the uterus and catarrhal erosions of the os uteri, whose cardialgic attacks recurred regularly every four weeks and lasted three days; but during the intervals they only appeared when leeches were applied to the cervix uteri, and they only lasted during the time of the application.

3. In other cases, nervous cardialgia depends on diseases of the spinal marrow or brain; and from analogy with other neuroses, it is probable, although it has not been proved by observations, that it may be caused by organic changes in the pneumogastric, or sympathetic nerves, swelling of their neurilemma, or tumors pressing on them.

4. Cardialgia may depend on dyscrasia. Infection of the blood, with malarial poison, occasionally seems to excite spasm of the stomach instead of the paroxysm of intermittent fever. *Romberg* attaches particular importance to arthritis as a cause, and in his own first attack of gout he suffered severely from spasm of the stomach.

5. Finally, we frequently cannot find, either during life, or on autopsy, any cause for cardialgia that has existed for years.

From the description above given, the attacks of pain caused by certain contents of the stomach, without any structural change, must be considered as nervous cardialgia. Among these are the cases of spasm of the stomach induced by excessive acidity, by the presence of worms in the stomach, by the exhibition of certain medicines, and occasionally after a cold drink and similar causes.

SYMPTOMS AND COURSE.—Like most neuroses, nervous cardialgia is distinguished from other diseases by its typical course, i. e., after intervals of freedom, follow paroxysms of the severest pain. Occasionally there is a regular type, so that the attacks recur at the same hour daily, or every second or third day.

It is impossible to describe a cardialgic attack more strikingly, or briefly, than has been done by *Romberg.*

"Suddenly, or after a precedent feeling of pressure, there is severe, griping pain in the pit of the stomach, usually extending to the back, with a feeling of faintness, shrunken countenance, cold hands and feet, and small intermittent pulse. The pain becomes so excessive that the patient cries out. The epigastrium is either puffed out, like a ball, or, as is more frequently the case, retracted, with tension of the abdominal walls. There is often pulsation in the epigastrium. External pressure is well borne, and not unfrequently the patient presses the pit of the stomach against some firm substance, or compresses it with his hands. Sympathetic pains often occur in the thorax under the sternum, in the œsophageal branches of the pneumogastric, while they are rare in the exterior of the body."

"The attack lasts from a few minutes to half an hour; then the pain gradually subsides, leaving the patient much exhausted; or else it ceases suddenly with eructation of gas or watery fluid, with vomiting, with a gentle soft perspiration, or with the passage of reddish urine."

Besides these severe attacks, we often see painful sensations, of various varieties and degrees of intensity, in the stomach, which also alternate with intervals of rest and freedom from pain, which are lessened, not increased, by external pressure, or by the introduction of food; these also are accompanied by sympathetic pain in the breast and back, reflex motions of the abdominal muscles, etc. It is these mild attacks, without "the feeling of faintness and impending death," that *Romberg* styles neuralgia of the pneumogastric, in contradistinction to neuralgia cœliaca."

DIAGNOSIS.—The character of the pain gives no aid in distinguishing cardialgic attacks, accompanying an ulcer of the stomach, from those due to neuralgia of the gastric nerves. In the former we also see the extension of the pain to the back and breast, and see them subside with vomiting and eructation, and the depressing effect of the pain on the patient. The following factors are important in judging between the two states: 1. In most cases, pains induced by ulcer of the stomach are increased by external pressure by introduction of food ("pressure from within"); while, on the other hand, nervous cardialgia is usually relieved by pressure over the stomach, and by eating. 2. In chronic ulcer of the stomach, dyspepsia and other symptoms of disturbance of the functions of the stomach are present during the intervals; these do not appear in nervous cardialgia. In accordance with this, nutrition is little impaired in the latter disease, and, when the neuralgia is not due to anæmia, the patient may look strong and healthy. 3. Dysmenorrhœa, metrorrhagia, sterility, and other symptoms which betray affection of the sexual organs, as well as decided chlorosis, render it probable that the affection is nervous in character; but too much weight must not be attached to these symptoms, for it is in just such cases that ulcer of the stomach is apt to occur. 4. The simultaneous occurrence of other neuralgic affections speaks for a similar nature in these attacks of pain. 5. Finally, genuine neuralgia of the stomach is excited by unknown causes, and often occurs while the stomach is empty; the attacks of pain in ulcer of the stomach almost always come after eating.

PROGNOSIS.—The prognosis is favorable in cardialgia dependent on poverty of the blood, which is not due to cancer, tuberculosis, or some other incurable disease. Those cases, also, that are caused by uterine complaints disappear with the cure of the original disease, if this be amenable to treatment. The prognosis is generally favorable, also, in those cases resulting from the influence of malaria or arthritis. On the other hand, treatment is almost always unavailing in the cases depending on affections of the brain or spinal marrow, and in those arising from unknown causes.

TREATMENT.—The indications from the cause require the energetic and early employment of the preparations of iron in chlorotic and anæmic cases. It is a great error to delay the use of iron in the treatment of chlorosis until the stomach is prepared for it, and the cardialgic attacks have passed away. The dyspepsia and cardialgia of chlorotic patients do not yield sooner to any remedies than to those which improve the state of the blood. The springs of Pyrmont, Driburg, and Cudowa are wonderfully beneficial in this affection. Among the officinal preparations of iron, the best is the ferri carbonas saccharata (Br.). *Blaud's* pills are also an excellent prescription (see treatment of chlorosis). In hysterical cardialgia, applications of leeches to the os uteri, touching ulcers on it with nitrate of silver, and other treatment, which we shall learn when speaking of uterine diseases, may be indicated, and may have a striking effect. In cardialgia excited by malaria or arthritis, the fulfilment of the causal indications answers for the treatment of the original disease.

The indications from the disease are best answered by the narcotics, and, among these, acetate of morphia is preferable to the extracts of hyoscyamus, belladonna, etc., which have also been recommended. This remedy is usually given in combination with the so-called antispasmodics, particularly with valerian, asafœtida, and castoreum. Recently, a mixture of equal parts of tincture of nux vomica and tincture of castor (dose, 12 drops during the attack) has been much used, and apparently with good effect. Metallic remedies also, particularly nitrate of bismuth, nitrate of silver, cyanide of zinc, have been recommended in spasm of the stomach; however, as they are scarcely ever given alone, but are used in combination with narcotics, their efficacy is problematical. Finally, *Romberg* recommends aiding the treatment by applying belladonna or galbanum plasters over the stomach, or rubbing in a mixture of mixtura oleosa-balsamicæ (℥ j) with tinctura opii (ʒ ij).

## CHAPTER X.

### DYSPEPSIA.

In the preceding chapters we have often spoken of dyspeptic symptoms, i. e., of signs of impaired digestion. Hence, while giving a separate chapter to dyspepsia, we shall only speak of those disturbances of digestion which arise without perceptible change of structure of the stomach. The different forms of this dyspepsia may be included under two heads: the digestion is impaired either because the gastric juice secreted is of abnormal quality, or because the movements of the stomach are diminished, and, consequently, the ingesta are not sufficiently mixed with the gastric juice. Digestion, which is a purely chemical process, can only be influenced by the nerves when they modify the secretions, or the movements of the stomach, and only in this sense is it proper to speak of nervous dyspepsia.

The change in the gastric juice may be either qualitative or quantitative. We know very little about the *qualitative* changes. They may consist in alteration of the proportion of the normal constituents to each other; thus we know that too slight an amount of free acid weakens the solvent power of the gastric juice; or in the fact that foreign substances are mixed with the gastric juice, such as urea in cases of retention of urine; or because, under certain circumstances, the constitution of the gastric juice is totally changed, some constituents being added and others disappearing. The symptoms caused by qualitative changes of the gastric juice are entirely unknown, and still less do we know the remedies for treating the state in question.

As to the *quantitative* changes of the gastric juice, the very unsuitable name of "atonic indigestion" has been given to the symptoms induced, where the gastric juice is insufficient, or where it is not sufficiently concentrated. In the etiology of gastric catarrh we mentioned that too scanty a secretion, or too poor a quality of the gastric juice, existed in anæmic and chlorotic persons. We there explained that this anomaly increased the inclination to chronic catarrh of the stomach, because, as the ingesta readily decomposed, their products excited intense irritation of the mucous membrane. To what has been said, we must add that the gastric mucous membrane is not affected in all the cases where the contents are abnormally decomposed, and that the cases where this membrane remains healthy should be carefully distinguished from those where it becomes diseased. The symptoms induced by restricted secretion of gastric juice are, it is true, frequently similar to those occurring in chronic catarrh, and even to those of chronic ulcer of the stomach. In this form of dyspepsia, also, the appetite is less, or is satisfied after eating very little. After eating, the epigastrium swells, and there is eructation of gases, or sour and rancid liquids; the patients suffer from flatulence, and are disturbed and anxious about their condition. Besides the nervous cardialgia occurring in anæmic and chlorotic patients, the excessive formation of acid may cause griping pain in the stomach (in the substances vomited by chlorotic patients, *Frerichs* found acetic acid and quantities of yeast fungus), and these cases may very readily be mistaken for chronic ulcer of the stomach. The diagnosis of this form of dyspepsia depends chiefly on the etiology. If the symptoms occur in chlorotic girls, about the age of puberty, or in persons weakened by venereal excesses, particularly onanism, or in those exhausted by care and anxiety, by continued work, or night watching, or if they come during convalescence, from protracted and exhausting diseases, and particularly if we can find that the nutrition was impaired before the appearance of the digestive difficulties, the chances are in favor of its being the so-called atonic dyspepsia, and against the existence of structural change of the stomach.

The condition of the tongue gives another point in diagnosis. While in chronic gastric catarrh there is a coated tongue and other signs of oral catarrh, in the dyspepsia of anæmic patients the tongue is usually clean, the taste unchanged, and there is no fetor from the mouth. In many cases the diagnosis is decided by the effect of fasting and of eating. Spiced and irritating substances, which increase the difficulty in chronic catarrh and chronic ulcer of the stomach, are well borne in atonic dyspepsia, and ease the painful symptoms which accompany it.

Above all, a mode of life that improves nutrition, the administration of iron, and sea-bathing, which have but little effect on chronic gastric catarrh, or chronic ulcer of the stomach, produce most brilliant results in dyspepsia dependent on anæmia or hydræmia. In some of these cases, particularly where the dyspepsia is accompanied by irritability or sensitiveness of the stomach, the pure bitters, such as quassia or hops, are very serviceable. We cannot explain the unmistakably favorable influence of these remedies on the gastric mucous membrane; for, while they are very irritant to the gustatory nerves, they have no effect when applied to the other mucous membranes, or to the skin. Quassia is generally ordered as a cold infusion. In the evening we may pour a cupful of cold water over about a teaspoonful of quassia-chips; by the next morning a bitter infusion will have formed, which is to be drunk fasting, or else water may be left for a while in a cup, turned from quassia-wood, and then drunk. Hop-bitter is generally used in the form of Bavarian beer, which is now brewed all over Germany; but we must take care that it comes from a trustworthy brewery, where, instead of hops, some injurious substitute is not used. The strong malt-extract, of which we previously spoke (page 246), has proved very efficient, in my hands, in several cases of dyspepsia, characterized by irritable indigestion. Occasionally it was almost the only nourishment the patients bore. It is not improbable that the preparations of nux vomica, which have a great reputation as stomachics, act like the above remedies, by their bitterness. The preparations most used in dyspepsia are the aqueous extract (gr. ½—1), alcoholic extract (gr. ¼—⅓), and the tincture (gtt. x—xii).

Too scanty a secretion of gastric juice, and the symptoms dependent on it, occur also in persons accustomed to great irritation of the stomach, as soon as they change their mode of life, and take their food without the addition of any stimulants. Unable as we are to explain how an organ becomes accustomed to irritation, there is no doubt of the fact. We may aptly compare the gastric mucous membrane of persons who daily use quantities of pepper, mustard, and other spices, to the nasal mucous membrane of habitual snuff-takers. In most persons small quantities of snuff excite great reflex symptoms, when taken into the nose, while the habitual snuff-taker can fill the nose with snuff without sneezing. Moreover, the secretion of gastric juice must be regarded as a reflex symptom, excited by the irritation of the ingesta on the mucous membrane. In the persons in question, the irritation from ordinary food is insufficient to produce a sufficient supply of gastric juice. Part of the ingesta remain undigested; it is decomposed, and we have the symptoms above described. If, on the other hand, the food be taken with a strong addition of spices, the patients do quite well, their nutrition goes on all right, and there is nothing to induce us to think that they have chronic gastric catarrh, or other organic change of the stomach, till finally symptoms occur which prove that the stomach has not borne these insults without injury. We must go very carefully to work in the treatment of these patients; we cannot allow them to retain their bad habits, but we should only break them off gradually. If we break either of these precepts, we may readily induce gastric catarrh or some other disease of the stomach.

Among the stomachics, which are indicated in the last-described form of dyspepsia (well called "torpid indigestion"), rhubarb is the best; it is giveh as powder or pills, or as the aqueous tincture (ʒj), but best as the vinous tincture (gtt. xx—xxx). In torpid dyspepsia, ipecac. (gr. ¼—½) has also a good reputation, particularly with English physicians. Finally, the bitter medicines containing an ethereal oil are good in torpid indigestion; among the most popular remedies of this class is elixir aurantiorum comp. (gtt. xxx—xl).

The dyspepsia of old persons also appears to be caused by too scanty a secretion of gastric juice, which partly depends on a lack of the materials necessary to its formation, and partly on diminished excitability of the gastric nerves. It is difficult to decide how far the disturbance of digestion in this, as well as in the first-mentioned form of dyspepsia, depends on bad nutrition of the muscles of the stomach; and it is sufficient to call attention to the fact that the retarded movements of the stomach consequent on this deficient nutrition may lead to incomplete mixture of the ingesta with the gastric juice, and hence to dyspepsia.

Abnormally-increased secretion of gastric juice does not cause dyspepsia, it is true; nevertheless, we will here relate the symptoms that it appears to excite, particularly when the stomach is empty. Vomiting is seen to result from irritations which do not affect the walls of the stomach itself, but neighboring organs, particularly the ureters or the ductus choledochus, or even distant organs, as the uterus. This is usually considered as depending simply on reflex movements. *Budd*, however, in a spirited and striking manner, calls attention to the fact that in such cases the nerves causing secretion of the gastric juice have become more active from the reflex irritation. When *Spallanzani* induced vomiting in himself before breakfast, by tickling the fauces, he threw up an acid fluid, which dissolved meat; this shows that mechanical irritation of the fauces may excite secretion of gastric juice, even when the stomach is empty. *Budd* further says that, where there is impaction of biliary or urinary calculi, the vomited masses are often very acid, even when the stomach was previously quite empty of food, and that the acid they contained was found by *Prout* to be muriatic. This circumstance and the decided and rapid removal of these gastric difficulties by alkaline remedies render it probable to him that part of the pain, and perhaps also the vomiting, depended on the irritation of the gastric mucous membrane by the juice poured into the empty stomach. At all events, the urgent advice of *Budd* and *Prout*, to give large doses of bicarbonate of soda (ʒ ij in a pint of warm water), is worthy of attention.

A number of renowned physicians, particularly in England and France, maintain the opinion that the occurrence of oxalic acid in the blood, along with some other symptoms of disease, results in a peculiar form of dyspepsia, which can only be cured by removing the oxalic diathesis. As the doctrine of the *oxalic diathesis*, and of the dyspepsia caused by it, has lately found many supporters as well as many opponents in Germany also, I will briefly state my position in regard to this still debatable question.

*Traces* of oxalate of lime are so often found in the urine of healthy persons, that it forms, as it were, a transition from the normal to the abnormal constituents of urine.

Quantities of this salt are found in the urine when the affected persons have eaten substances containing oxalates, such as certain vegetables, sorrel, sheep-sorrel, or rhubarb-stalks; they also occur temporarily after the free use of carbonated drinks, such as champagne, seltzer-water, soda-water, etc. In all of these cases no disturbance of digestion or of the general health is observed.

It is different in those cases where large quantities of oxalate of lime occur for a length of time in the urine; here there are almost always other morbid symptoms. Sometimes, along with the oxalate of lime, we find spermatozoa and quantities of mucus in the urine, which render it probable that, in these cases, the oxalate of lime is not excreted by the kidneys, but forms in the urine during its stay in the urinary passages. Since *Gallois* and *Hoppe-Seyler* have shown that the characteristic crystals of oxalate of lime (octohedrons, so-called envelope-shaped) increase in size after the urine stands awhile, we cannot doubt that this salt probably also forms in secreted urine from the decomposition of mucus. We must dismiss the idea that the insoluble salt formed in the urinary passages can have an injurious influence on the stomach and the rest of the organism; then the symptoms of this form of oxaluria, disturbance of the general health, melancholia, paleness, etc., will be naturally explained by the coincident spermatorrhœa and the catarrh of the urinary passages. But, finally, there are a greater number of cases where the oxaluria cannot be deduced from a decomposition of the secreted urine, but we are obliged to refer the presence of the oxalates in the urine to an increased formation of oxalic acid in the blood, that is, to an oxalic diathesis. Now, what causes the proportionately rich formation of this substance in the blood and its proportionate abundance in the excrements of the body, where, normally, only traces of it can be found? At present, this question cannot be satisfactorily answered. However, there is no doubt that oxaluria is more frequent in England, where the people eat and drink more and better food and liquor than in Germany, and that in the latter country it is almost only seen among people of the better classes, who enjoy the pleasures of the table. Little inclination as I have for chemico-physiological hypotheses, I still believe that, from these facts, we may consider it as probable that the oxalic diathesis and oxaluria are to some extent caused by the supply of nutriment to the body exceeding the requirements. At the same time, I will not dwell on the question as to whether, while this misproportion exists, the consumed products can only be brought to a low degree of oxidation; or whether the abnormal increase of substances at a low grade of oxidation in the excrements of the body, such as oxalic acid, uric acid, etc., depends upon other complicated and still wholly unknown causes. I think I can support the observation that, in general, persons who become fat with good living remain healthier than those who produce but little fat under like circumstances, and particularly more so than those who, continuing their mode of life, lose fat. While, as a rule, the former only suffer from certain inconveniences dependent on their corpulence, the latter often complain of all kinds of distresses, which physicians usually associate with portal obstructions or hæmorrhoids, or deduce from gouty, rheumatic, or catarrhal diseases. This renders it probable that, in many cases where there is the above-mentioned disproportion between the supply and demand, and this is not at once removed by increased production of fat, the products of the change of tissue are modified, and that the above-described difficulties depend on an abnormal nutrition of the different organs by the blood, which is overloaded with quantitatively or qualitatively abnormal excremental material. After a long continuance of the hypochondriasis, the disturbance of digestion, the pharyngeal and bronchial catarrh, pain in the joints, especially the smaller ones (of which groups of symptoms, first one, then another becomes prominent, or is even exclusively present), such persons usually become feeble, pale, and thin, so that they appear to have a severe and grave affection. The urine, which is usually concentrated and acid, does not always show characteristic changes. But, in most cases, abundant sediments of uric-acid salts are occasionally deposited. According to my experience, tonic treatment, and the use of wine, and preparations of quinine and iron, to which we may be tempted by the weakness, pallidity, and emaciation of the patient, are almost always injurious; while the use of alkaline-saline mineral waters has the happiest results, particularly when combined with cold washing, or cold douches (as is often done by Dr. *Müller*, in Homburg), or if sea-bathing be tried after the water-treatment. I have no great experience in oxaluria and the oxalic diathesis, but those cases that I have met resembled most closely those I have just described, although I will not consider them as exactly identical; they had the same etiology, the same varied complaints (not exactly corresponding to any of the usually-described forms of disease), the same sleepiness, paleness, and emaciation, only the urine, which is usually acid, contained no sediment of urates, but had crystals of oxalate of lime. Hence I deem it most proper to regard the dyspepsia, which occurs as one of the many symptoms of oxaluria, as the result of a constitutional derangement, and that this derangement is developed in persons predisposed to it, by the manner of living above described. At present we have no idea which link in the long chain of processes between assimilation of nourishment and the excretion of the used-up constituents of the body, is first changed by this injurious influence, which induces the formation of quantitatively or qualitatively abnormal products.

In this affection we should employ the treatment which I above recommended for the diseases allied to, if not identical with, the oxalic diathesis. (The occurrence of oxalate of lime, as a final product of the change of tissue in the oxalic diathesis *alone*, is opposed to their complete identity.) The administration of nitric acid (twenty drops of the dilute acid two or three times daily), recommended by English physicians for the oxalic diathesis, and the forbidding of all saccharine articles of food, appear to depend more on theory than on the results of practical experience.

Before closing the consideration of dyspepsia, I wish to speak of a peculiar form of dizziness which is a very frequent but inexplicable symptom. *Trousseau*, who considered that it arose from the dyspepsia, called it *vertige stomacale*. Almost any practitioner can refer to some case among his patients that will correspond with the true and life-like description given by *Trousseau* of the vertige stomacale, the vertigo a stomacho laeso, or, as our people call it, abdominal dizziness (bauchschwindels). The disease, which subsequently becomes very obstinate and tedious, usually begins acutely without any premonition. The patient, who just previously felt perfectly well, complains of great dizziness; it seems to him as if every thing around him, or as if he himself, were whirling or rolling about. Besides this hallucination, there are usually abnormal sensations in the head, which, the patient says, he cannot call pain, but which he in vain attempts to describe. Sometimes patients say their heads feel "empty," or "light;" others speak of a "numbness," of a "sensation of undefined pressure," of a "cloud arising in the head;" besides this, there are usually sparks before the eyes, noises in the ears; the patients are afraid of falling, seek support, want to sit down or lie down. These attacks, during which the color is either unchanged or becomes pale, usually pass off after a few minutes; but, while they last, they greatly terrify the patients and those around them. They often, but not always, terminate with gaping and eructations.

Sometimes there is only one such attack, but more frequently there is a recurrence, sooner or later. It is, at the same time, very remarkable that the new attacks are excited by apparently insignificant causes, such as walking on a polished floor, or a smooth sidewalk, or by passing a grating; also, that on such occasions the patient does not become dizzy if he holds the hand of even a small child, or rests on a slender cane; lastly, that there is no dizziness if he is engaged in something that occupies his whole attention, or if he be mentally excited. I knew one patient who could on no consideration walk alone through a hall, or across an open square, while he could dance without trouble in the same hall, and unconcernedly rode a spirited horse across the same square. The longer the affection lasts, the more the thoughts of the patient are directed to his enigmatical and curious state. He becomes greatly depressed by the idea that he has disease of the brain, and particularly when he hears that dizziness was one symptom in some other patient who actually had cerebral disease, who perhaps died of softening of the brain, that most terrible bugbear of the laity. Physicians also are often mistaken, and order bleeding, derivatives, preparations of iodine, saline springs, and forbid wine and beer, and restrict the diet as much as possible. If this treatment be ineffectual, and under it the patient becomes pale and thin, the physician often changes his opinion: he suspects that the dizziness is caused by anæmia of the brain; then he prescribes iron, advises the use of wine and beer, and places the patient on a nutritious animal diet. However, this treatment also proves unavailing, and the patient returns unimproved from the Alps, from the cold-water cures, and from the sea-shore. As above mentioned, *Trousseau* believes that these attacks of dizziness depend on dyspepsia; at the same time he acknowledges that in many cases the signs of dyspepsia are so slight as to be readily overlooked. He relates cases of the successful treatment of "vertige stomacale" by the alternate administration of infusion of quassia and a composition of carbonates of the alkalies. Even from this prescription I have never seen any benefit; and while I must acknowledge that the first attacks of dizziness, affecting the patients that I have seen, usually occurred after an indigestion, and were accompanied by dyspeptic symptoms, still, in none of them were there evident signs of indigestion during the subsequent attacks, which often continued for years. I believe the repetition of the attacks of dizziness to be due to psychical causes. As there are persons who become dizzy when they stand on the edge of a precipice, or on a high tower; and as one who has once become dizzy on such an occasion, is almost certain to become so again in a similar position; so, a person, who has once become dizzy in his chamber, or while walking over an open square, is in the greatest danger of becoming so again on a similar occasion. *The fear of the dizziness* is a strong predisposing cause for it in this form, just as it is in that where people have it from standing on a high place. On the other hand, concentrated attention on any point, mental emotion, or even a slight noise, may, to a certain extent, prevent attacks from either cause. As a proof of the correctness of this view, I may mention the case of a clergyman, who, while going to the pulpit in his church, had a severe attack of dizziness, and fell to the floor. For years, as long as I had a chance to observe him, this patient never had another attack of severe dizziness; but he never entered his pulpit after the first attack. On two or three attempts, he thought he noticed premonitions of the dizziness, which induced him to give up further attempts; he had to give up his employment, just as a tower-keeper or a roofer would have to abandon his, if, while engaged in his avocation, he had one or two severe attacks of dizziness.

# SECTION V.

## *AFFECTIONS OF THE INTESTINAL CANAL.*

### CHAPTER I.

### CATARRHAL INFLAMMATION OF THE INTESTINAL MUCOUS MEMBRANE—ENTERITIS CATARRHALIS, CATARRHUS INTESTINALIS.

ETIOLOGY.—In the mucous membrane of the intestines also catarrh is the constant result of every hyperæmia, whether the vascular fulness depend on purely mechanical causes, or on other injurious influences. At the commencement of the disease, and in acute cases, the hyperæmia induces more particularly extensive transudations of a salty fluid, deficient in albumen; subsequently, and in chronic cases, on the other hand, it generally leads to abnormal production of mucus and cells.

Intestinal catarrh, and particularly the chronic variety, is one of the most frequent of diseases:

1. It constantly accompanies obstruction of the circulation of the liver. The impeded escape of the blood from the portal vein must necessarily cause overfilling of the intestinal veins, and so produce catarrh of the intestines.

2. It frequently, but less constantly, accompanies the diseases of the respiratory and circulatory organs, which cause obstruction to the evacuation of the vena cava. As in these affections there is venous congestion throughout the circulation, it also occurs in the intestinal mucous membrane; in these cases the hyperæmia and catarrh of the intestine represent, as it were, the cyanosis of the skin.

3. More rarely, disturbance of the external circulation appears to cause active hyperæmia and catarrh of the intestines. In this class appear to belong the excessive hyperæmia of the intestines, which accompany severe inflammation of the skin, caused by burns, as well as the evanescent hyperæmia with copious serous exudation induced by sudden exposure of the skin to low temperature, as by travelling in the mountains (*Bidder* and *Schmidt*). We will not attempt to say

whether cases of catarrh caused by coldness of the feet and of the lower part of the body, which depends on the continued action of cold, and the chronic cases induced by damp, cold climates, belong in this class.

4. The severe intestinal catarrh frequently occurring in peritonitis, particularly puerperal peritonitis, must also be considered as due to excessive active hyperæmia. In these cases the intense inflammation leads to œdema of the subserous tissue, of the muscular coat, and of the intestinal mucous membrane. We see similar œdema occur in the vicinity of all inflammatory disturbances of circulation, and we have repeatedly described it as collateral œdema, or œdema from collateral fluxion. It readily explains the watery passages, which often accompany peritonitis, in spite of the paralysis of the muscular coat of the intestines.

5. Fluxion to the intestinal capillaries with consecutive serous transudation appears also to be the cause of diarrhœa induced by mental excitement. In these cases we must suppose that the afferent vessels are dilated by nervous influence, and this hypothesis has at least received some support, since *Budge* showed that there is constant diarrhœa after extirpation of the cœliac ganglion in rabbits.

6. In most cases hyperæmia and catarrh of the intestinal mucous membrane are the results of *local irritation*. Most purgatives act in this way, for very few of them purge, by acting as concentrated solutions of salt, i. e., by endosmotically inducing a copious flow of liquid from the intestinal vessels into the intestines, without exciting hyperæmia. Catarrh of the intestines is caused much less frequently than was formerly supposed by large quantities of bile, and not very often by the presence of parasites. In this class belong the cases of intestinal catarrh occurring after the use of certain non-medicinal substances, such as some kinds of vegetables, but particularly those cases due to the passage of undigested and decomposing substances from the stomach into the intestines (see etiology of gastric catarrh). It is very frequently caused by the retention of fecal masses; if these remain for a length of time at any part of the intestines, they decompose, and form products which have a very injurious and irritant influence on the intestinal mucous membrane. To *Virchow* is due the credit of calling attention to the frequent occurrence of local peritonitis and the change of position, constriction, and twisting of the intestines dependent on it. Indeed, these are in many cases the causes of habitual constipation; and some chronic ailments, which in general terms are called "chronic abdominal difficulty," depend solely on distortion and constriction of the intestinal canal, on the development of gases from the decomposed fæces, or on consecutive intestinal catarrh.

7. At certain times intestinal catarrhs prevail from unknown causes, which, without our exactly understanding, are usually called *genius epidemicus gastricus.*

Finally, it is frequently only a symptom of a general disease. In the lower animals it may always be excited by the injection of putrid substances into the veins (*Stich*), it always accompanies typhoid fever, and is the severest symptom in Asiatic cholera. We shall hereafter speak of these symptomatic forms, as well as of those accompanying ulceration and degeneration of the intestinal canal.

ANATOMICAL APPEARANCES.—Catarrh rarely affects the entire intestinal canal. It is most frequent in the large intestine, less so in the ileum, and rarest in the jejunum and duodenum. The anatomical changes left in the cadaver by acute catarrh are sometimes pale, at others dark, redness, swelling, relaxation, and friability of the mucous membrane, which is sometimes diffuse, at others limited to the vicinity of the solitary glands, and of *Peyer's* patches, and a serous infiltration of the submucous tissue. Occasionally, after death, the injection has entirely disappeared, and the mucous membrane appears pale and bloodless. Swelling of the solitary glands and glands of *Peyer* is an almost constant appearance; they distinctly project above the surface of the mucous membrane. The mesenteric glands also are usually found hyperæmic and somewhat enlarged. The contents of the intestines consist at first of plentiful serous fluid, mixed with detached epithelial and young cells, subsequently of a cloudy mucus, which is adherent to the wall of the intestine, and contains epithelial structures.

In *chronic* intestinal catarrh the mucous membrane looks more brownish-red or slate-gray; it appears puffed up, and, particularly in the rectum, forms polypoid protrusions. The enlarged follicles usually project even more distinctly than in acute catarrh, as white nodules above the surface, covered with tough, gray, or puriform mucus. Sometimes, though more rarely than in chronic gastric catarrh, there is hypertrophy of the muscular coat, which may cause a constriction of the intestinal canal analogous to simple stricture of the pylorus.

In some cases catarrhal inflammation has a diphtheritic character. Then superficial sloughs form on the very red mucous membrane, so that it looks as if sprinkled with bran. After the sloughs have been thrown off, shallow erosions, which bleed readily, are left. This anatomical appearance, which is almost exclusively found at the lower part of the large intestine and in the rectum, and occurs there from a collection of hardened fæces at that place, answers to the clinical picture of a mild catarrhal dysentery.

The severe forms of intestinal catarrh may lead to ulceration; we may have either diffuse catarrhal or follicular ulcers in the intestine. Diffuse catarrhal ulcers result from acute or still more frequently from chronic inflammation, to which an acute attack supervenes. The most frequent cause is foreign bodies in the intestine or retained fæces. Hence they most frequently occur where the contents of the intestines are most readily arrested; in the cœcum, ascending colon (typhlitis stercoralis), processus vermiformis, then in the rectum and colon, above constricted or distorted places. The dark-red and swollen mucous membrane softens, and is destroyed by suppuration in its tissue; the result is a loss of substance which exposes the submucous or muscular tissue. If the ulcer heal at this stage, the loss of substance is filled with granulations, and a firm cicatrix remains, which almost always constricts the intestine. In other cases the muscular and serous coats are also destroyed and the intestine perforated. While the destruction proceeds from within outward, a partial peritonitis may occur, and cause a union with neighboring portions of intestine, thus preventing the escape of the contents of the bowels into the abdomen. This course is most frequently seen in perforation of the vermiform appendage. Perityphlitis (a phlegmonous inflammation of the loose connective tissue attaching the cœcum and ascending colon to the iliac fascia) occurs as frequently as peritonitis in inflammation and ulceration of the cœcum, which is called typhlitis stercoralis. As this may also occur independently of disease of the intestines, we will speak of it in a separate chapter.

The second form of catarrhal ulceration, the *follicular ulcer*, occurs almost exclusively in the large intestine, particularly at its lower part. It causes great destruction, and is characterized by the slight reaction shown by the mucous membrane, in the vicinity of the ulcer. According to *Rokitansky's* masterly description, it comes in this way: At first the follicles are greatly swollen, surrounded by a dark-red vascular ring; subsequently they ulcerate from within; the pus breaks through; there is a small, follicular abscess, which has red, spongy walls, and a small, ulcerated, finely-fringed opening. While the ulceration gradually destroys the whole follicle, the hyperæmia of the adjacent mucous membrane gradually disappears; the ulcer is then about the size of a lentil-seed, round or oval. The ulceration soon extends to the surrounding mucous membrane; the round form of the ulcer is lost; large, irregular ulcerations occur, or, for a considerable distance of the intestine only, some islands and irregular projections of the membrane are preserved, while elsewhere the submucous or the muscular tissue is exposed. In the intestine we usually find a grayish-red, half-fluid, floccular substance, mixed with undigested ingesta.

SYMPTOMS AND COURSE.—In acute intestinal catarrh, besides the serous transudation, there is acceleration of the movements of the intestines, so that the passages are not only more fluid, but they become more frequent. Diarrhœa, which is often preceded by rumbling in the intestines, is the most constant, and occasionally the only symptom of acute intestinal catarrh. Pain and other symptoms may be absent, the strength and nutrition of the patient may remain normal, if the evacuations be not too copious and long continued. In such cases the laity usually regard the diarrhœa as a favorable symptom, from which they anticipate a cleansing of the body and all sorts of benefit. At first the evacuations consist of thin fecal matters (diarrhœa stercoralis). If the serous transudation and the accelerated peristaltic movement continue after all the fæces present in the bowels have been evacuated, the dejections gradually lose the peculiar fecal odor, and consist of salty transudations mixed with epithelial masses (cylindrical epithelium), young cells, and more or less undigested and slightly-changed ingesta (diarrhœa serosa). The color of fluid stools is usually some shade of green; this does not depend on an abnormal quantity of bile being poured into the intestines, but on the bile being evacuated with the fluid and the intestinal secretions, before it has undergone the normal changes. The more copious the transudations, the paler they become, because the bile, mixed with them, is insufficient to color the whole. There is scarcely a trace of albumen in these catarrhal evacuations; but there are not unfrequently crystals of phosphate of magnesia and ammonia, whose presence was long considered as characteristic of typhous passages, and there is usually plenty of chloride of sodium. Generally, after the diarrhœa has lasted a day or two, or even longer, the normal transformations of the ingesta begin again; the evacuations become less frequent, and again acquire their feculent appearance and smell. A more or less obstinate constipation generally follows the diarrhœa.

In other cases, besides the diarrhœa, there are pains in the abdomen. These are chiefly periodical attacks of griping pain or colic, during which, if the pain be severe, the patient becomes very pale and cool. These colicky pains usually subside when a discharge from the bowels has just taken place, or is about to occur. A continued feeling of pressure or soreness, and of sensitiveness to pressure, in the abdomen, is seen far more rarely than the above-mentioned attacks of pain. It is only in the rare cases, where acute intestinal catarrh accompanies extensive burns of the skin, that the latter pains become very severe. This peculiarity and the presence of blood in the evacuations distinguish this form from all others.

In acute intestinal catarrh the abdomen is often somewhat prominent, and quantities of badly-smelling gases escape with the passages.

The development of gas in the intestines cannot be regarded as a symptom, or as the result of acute intestinal catarrh, as long as this is in the stage of moderate transudation and increased peristaltic motion; it more frequently depends on the same causes as the catarrh itself, particularly on the passage of undigested and decomposing food from the stomach into the intestines.

Finally, acute intestinal catarrh is not unfrequently accompanied by fever. If it was caused by catching cold, it generally shows the peculiarities of so-called catarrhal fever; in other cases the fever is more severe, and, if the stomach be affected at the same time, we have the symptoms of a gastric, bilious, or catarrhal fever, which we shall hereafter describe.

Acute intestinal catarrh runs the above course when it affects a large portion of the intestines, or if, as is usually the case, it be located in the lower part of the ileum and colon. Catarrh of the duodenum often accompanies catarrh of the stomach; but it can only be recognized when it extends to the ductus choledochus, and so causes obstruction of the gall-ducts and jaundice; in all other cases it modifies the symptoms of gastric catarrh too little to be recognized.

Catarrh of the small intestines may run its course without diarrhœa, if the fluid contents of the small intestine remain for some time in the large intestine, and become thickened by resorption of the watery portions. If, with the symptoms of gastric catarrh, we have loud gurgling and rumbling in the abdomen, showing that there are gases and liquids moving about in the bowels, but if the anticipated discharge do not occur, we are justified in supposing that the gastric catarrh has extended to the small intestines, but not to the large.

There is often catarrh of the lower part of the large intestine and of the rectum without coincident disease of the other parts of the intestinal canal. When the inflammation is very severe, and shows a change from the catarrhal to the diphtheritic form, the symptoms are peculiar. Just as in dysentery, the passages are preceded by severe cutting pains, which spread from the navel toward the sacrum. Then there are spasmodic contractions of the sphincter, burning sensations at the anus, and, with severe pressure and straining, more or less white and glassy mucus, often mixed with blood, is evacuated. After this there is generally relief for a while, when the pains begin again, and the scene is repeated. Occasionally masses of hard fæces are passed, and the patient is left at rest for some time. Under proper treatment, i. e., if we immediately remove the hardened fæces, which excite and maintain the disease, *catarrhal dysentery* (as this disease is properly called) may be quickly cured. Improperly treated, it may be protracted, and lead to follicular ulcerations.

Finally, if the acute catarrh be confined to the rectum, there is also a constant desire to go to stool; the passages are mucous, or bloody mucus without any fæces; but there are none of the characteristic pains in the belly that precede the stools in catarrhal dysentery.

In adults, chronic intestinal catarrh rarely leads to extensive serous transudations into the bowels; in most cases the secretion from the mucous membrane is scanty and mucous. Hence, in adults, this disease is rarely or only temporarily accompanied by diarrhœa; on the contrary, the patients are usually constipated. The tough mucous coating over the wall of the intestine hinders resorption, and interferes with the nutrition; patients become debilitated and emaciated, and their complexion assumes a pale or dirty-gray color. Moreover, the mucus in the intestines acts as a ferment on the other contents, inducing decomposition, thus setting free quantities of gas, which inflate the bowels, and cause great annoyance; the belly becomes tense, the diaphragm is pressed upward, the respiration impaired; compression of the arteries causes congestion of other organs, particularly of the brain. Under such circumstances the passage of flatus is a great event for the patient, and it affords him much gratification. Besides the habitual constipation, the disturbance of the nutrition, and the flatulence with its results, there is almost always great mental disturbance, like that which we have already described among the symptoms of chronic gastric catarrh. The patients either occupy themselves entirely with their physical state, and have no brains or time for any thing else, or they are subject to a total indifference and despair. In this connection it is well worth attention, that, on autopsy of lunatics and suicides, we often find flexions and abnormal positions of the intestines, which are the most frequent causes of chronic intestinal catarah. Occasionally the habitual constipation is temporarily interrupted by severe colicky pain and a diarrhœa, by which quantities of mucus and badly-smelling fæces are evacuated. As this interlude often occurs without perceptible cause, it appears as if the decomposition of the contents of the intestines occasionally formed products which were particularly injurious and irritating to the mucous membrane of the bowels, and increased the chronic to an acute catarrh. Chronic catarrhs, running the above course, are among the most frequent, troublesome, and obstinate of diseases. From the inefficacy of the remedies prescribed, many patients despair of medical aid, and fall into the hands of charlatans, or use *Morrison's* pills, *Léroi's* herbs, *Strahl's* pills, or other domestic remedies. We shall hereafter show that these remedies, being laxative, undoubtedly have a favorable influence on the difficulties that accompany chronic intestinal catarrh, and that they owe their reputation as universal remedies to the great frequency of this affection.

Occasionally, however, chronic intestinal catarrh is accompanied by increased secretion from the mucous membrane and accelerated peristaltic movement of the bowels, and then runs its course as chronic diarrhœa. But in adults these cases are very rare; hence, diarrhœa, lasting a week or a month, must always excite the suspicion that there are more severe lesions of the intestines, and we should not consider simple catarrh as the cause of such cases till we have excluded other lesions. In such cases the dejections consist of quantities of glairy or puriform mucus, mixed sometimes with softened fæces, or sometimes with undigested food, if the catarrh be very extensive (diarrhœa lienterica). If colorless masses of mucus or puriform fluids be passed at some times, while at others hard scybola are evacuated, we may decide that the lower part of the large intestine is the seat of the disease, and that there is danger of the catarrh passing into follicular ulceration. Occasionally the diarrhœa ceases for a few days, giving place to obstruction, and then begins again more severely. Sometimes patients die of exhaustion from the chronic diarrhœa; but then we usually find some further disease or change in the intestines.

The case is quite different in the chronic intestinal catarrh of *children*. This almost always runs its course as an obstinate and exhausting diarrhœa, and we must be careful not too hastily to diagnosticate tuberculosis of the intestines, or mesentery, or a catarrhal ulcer, from this symptom. In the intestines of most children who die of chronic diarrhœa, usually with the imperfect diagnosis of "marasmus," on *post-mortem* examination, we find only the traces of a chronic catarrh, which may readily escape notice. This disease most frequently occurs toward the end of the first year, shortly after weaning (diarrhœa ablactatorum). At first the evacuations are more mucous and less copious than natural, have an acid reaction, and either immediately after being passed, or after being exposed to the air for a while, they have a greenish color. This depends on the admixture of unchanged bile, and on higher oxidation of the still retained coloring matter of the bile. Subsequently the dejections are very copious, watery, occasionally clay-colored, fetid, and mixed with undigested food. The previously healthy, well-nourished child is at first but little affected by this diarrhœa, but some fatal judgment often asserts it to be a safety-valve that protects the child from convulsions during teething, and that must not be stopped. Hence it often happens that the doctor is not called till the child has become flabby and relaxed, and then it is frequently difficult to master the disease; the diarrhœa continues, the child emaciates more and more; and a large number of children die during their second year from chronic catarrh of the intestines. In babies put out to board, chronic intestinal catarrh usually appears earlier and runs its course quicker. The mother of such a child, which has previously been healthy and plump, and whose appearance was the best recommendation for the fitness of the mother as a nurse, often takes a position as wet nurse even six or eight weeks after her confinement. The child is given over to some old woman, who feeds him with bad milk and spoiled pap, and, to prevent his crying too much, gives him a sugar teat or crust of bread during the intervals between meals. Diarrhœa soon occurs, emaciation goes on rapidly, and soon becomes extreme; fat and muscles disappear, the child becomes wrinkled, and looks like a little old woman; his flabby skin flaps about him like a loose pair of trousers; there are excoriations about the anus, and the oral mucous membrane is covered with thrush deposit. While the child which the nurse suckles flourishes, her own child usually wastes away and dies in the third or fourth month. In large cities, women who gain a living by taking children to board bury three or four, or even more, every year. Even in these cases, on autopsy, nothing is usually found but the signs of excessive wasting, and the slight remains of chronic intestinal catarrh. The latter may be considered, in the diarrhœa ablactatorum, as a series of daily returning acute catarrhs, which are daily excited by the passage of undigested and decomposed ingesta into the intestines.

We shall next speak of the most frequent form of the severe catarrhal inflammations that lead to ulceration of the mucous membrane, and not unfrequently of the entire wall of the intestine, viz., *typhlitis*, or, as it is usually called, *typhlitis stercoralis*. Sometimes there are premonitory symptoms, before attaining the stage of severe inflammation, that we call typhlitis; collections of fæces in the cœcum and ascending colon cause repeated attacks of colic and catarrh; so that from time to time the patient complains of stomach-ache, and has alternate constipation and diarrhœa. In other cases there are no premonitions, and even the first retention of fæces in the cœcum or ascending colon leads to severe inflammation and ulceration of the wall of the intestine. When this occurs, the muscular coat loses its power of contraction, and there is almost as great obstruction to the advance of the contents of the intestines as there is in constriction or adhesions of the bowels. Mucous or bloody mucous masses, the result of catarrh in the lower portion of the rectum, are passed, but there is no proper defecation. The contents of the small intestine cannot pass downward, hence are driven upward by the contractions of the intestinal muscles; there are so-called anti-peristaltic movements. The contents of the small intestines entering the stomach cause severe irritation; nausea and vomiting occur; at first the food in the stomach is vomited, then green, bitter, bilious masses, and in rare cases a brownish fluid of disagreeable taste and feculent odor (*Ileus*, *miserere*). From these symptoms, we may be certain that an obstruction to the progress of the contents of the intestines has occurred at some point; in the few cases where the pains in the right iliac fossa are slight, and when no tumor can be found there, we may be unable to determine the nature of this obstruction; but, in most cases, besides the constipation, there are severe pain and a characteristic tumor, which put an end to all doubt. The pains spread over the right lower part of the abdomen are marked by severe exacerbations, with intervals of comparative ease, and are increased by the slightest pressure in this region, as well as by every motion. On palpation, which the patients usually fear greatly, we feel a tumor, which has a sausage-like shape, and extends from the right iliac fossa toward the lower margin of the ribs. This tumor corresponds so exactly to the shape and position of the cœcum and ascending colon that it may be readily distinguished. Improvement begins in the above stage in favorable cases; the patient has several passages, with severe griping pains in the bowels, large masses of badly-smelling fæces are evacuated; vomiting subsides, the tumor decreases and disappears gradually, as only part of it is due to the contents of the intestine, the rest depending on the swelling of the wall of the intestine. But the disease does not always take this favorable course; on the contrary, in most cases, the inflammation extends from the serous covering of the cœcum and ascending colon to the peritonæum of the neighboring intestine and abdominal wall, and to the connective tissue uniting the ascending colon to the iliac fascia. From the extension of the peritonitis, the abdominal tenderness becomes more diffuse, the swelling loses its peculiar sausage shape and grows broader; from the perityphlitis (inflammation of the connective tissue behind the colon), there are pains in the right thigh, or a feeling of numbness; the psoas and iliacus muscles are infiltrated and cannot contract, so that the patient cannot raise his thigh. In these cases, the patient usually lies on the right side with the body bent forward, and dreads every movement, for in this position the abdominal muscles are less tense, and the psoas and iliacus are most relaxed. With the above symptoms the disease has not unfrequently attained its acme, and now gradually improves. As the typhlitis disappears, the secondary inflammations cease, and the exudation is gradually absorbed. In such cases, the pain in the abdomen subsides; the tumor, which had been regularly advancing toward the median line of the body, again becomes smaller, and finally disappears altogether. In the same way the pain and feeling of numbness in the right thigh pass off, the psoas and iliacus may again be contracted, and the thigh again raised. In unfavorable cases, the inflammation gradually affects the whole peritonæum or the incapsulated exudation is not absorbed, but keeps up a chronic peritonitis, and the patient succumbs to the protracted fever accompanying this disease. Finally, the walls of incapsulated exudation may gradually ulcerate, and there may be perforation outwardly, into neighboring parts of the intestines, or into other organs; we shall enter into this more particularly when speaking of peritonitis. Bad termination of the peritonitis, particularly its rapid spread over the entire peritonæum, should excite the suspicion that the ulceration of the cœcum has led to perforation; however, perforation is quite rare in this form, and there are very few cases where it can be certainly recognized during life. When perityphlitis results in formation of abscess and burrowing of the pus, its terminations are very varied (see Chapter VI.).

Ulceration of the processus vermiformis is usually accompanied by pain in the right iliac region; but this is usually so undecided that it is almost always impossible to interpret it correctly. It is not till the ulceration has reached the peritonæum, or when this has been destroyed and the above-described symptoms of partial peritonitis or those of perityphlitis occur, that the disease can be recognized. We could not at all determine whether the vermiform process or the cœcum was the starting-point of the consecutive inflammations, were it not for the absence of premonitory symptoms and of the obstruction and vomiting, but particularly of the characteristic tumor. If we are called to a case in a patient where there is already extensive peritonitis or formation of pus as a result of advanced perityphlitis, and who can give only a very incomplete history of his disease, the two affections cannot be distinguished. Although the peritonitis and perityphlitis in ulceration of the processus vermiformis are in most cases caused by its perforation and escape of its contents, these diseases may nevertheless run the above-described favorable course. This is most frequently the case when the perforation takes place gradually, so that the intestine becomes attached to the surrounding parts and the rest of the peritonæum is thus protected from injury by the escaping contents. Finally, firm adhesions may form; the pus and escaped masses may be incapsulated in a dense tissue, or they may perforate outwardly, while the point of perforation in the processus vermiformis is closed by a dense cicatricial tissue, so that no further escape takes place.

Severe inflammations and ulcerations of the intestines at other points than those above mentioned are much more rare; their most frequent seats, however, are in the transverse colon and sigmoid flexure. symptoms are similar to those of typhlitis, and consist in obstinate stipation, pain in a circumscribed spot in the abdomen, and the occurrence of the characteristic tumor. They very rarely induce general peritonitis, as it is much easier to remove the collections of fæces at these points and thus arrest the inflammation.

Follicular ulcers of the intestines are most frequently met in cachectic individuals. At first the symptoms are those of protracted catarrh of the large intestine; but we soon find peculiar translucent lumps, resembling swelled sago, in the mucous, white, and transparent masses, whose passage is preceded by slight tormina, and accompanied by moderate tenesmus. Occasionally there are passages of fæces with whitish or bloody mucus and the sago-like lumps. The mucous masses now become more opaque, fluid, yellowish white, and purulent, and we have the form of diarrhœa that was formerly called fluxus cœliacus, or diarrhœa chylosa. In this stage, also, the passages may sometimes be of normal color and consistence. If the follicular ulcers heal, strictures almost always result, and hence there are obstinate constipation, great inclination to flatulence, and the above-described symptoms of chronic catarrh.

Diagnosis.—Acute intestinal catarrh, unaccompanied by fever, is not readily mistaken for other diseases.

We shall hereafter speak of the diagnosis of idiopathic gastric and intestinal catarrh, occurring in the commencing stage of typhoid fever.

Those cases of chronic intestinal catarrh, where constipation, flatulence, and mental disturbance are the prominent symptoms, are often mistaken. Not long since it was almost universally believed that these symptoms depended mostly on disease of the large abdominal glands, particularly of the liver. The patients were sent to Karlsbad to be cured of their "biliousness," and, when they returned improved, it was considered as an evidence that the diagnosis had been correct. After accurate and unprejudiced autopsies had shown that chronic abdominal derangements, as the above symptoms were characterized, were rarely caused by perceptible changes of the liver, spleen, or pancreas, and, on the other hand, that great degeneration of these organs, as found on autopsy, had not always been accompanied during life by severe indigestion, a new error crept in. Many physicians, with *Rademacher*, considered it as proved that there are numerous diseases of the liver, spleen, and pancreas, which leave no perceptible changes of structure. It is unnecessary to enter into an argument against such a hypothesis, and we shall only call attention to the unheard-of method by which these diseases of the liver, spleen, pancreas, etc., are diagnosticated. If an affection, which, according to our physiological knowledge, has not the most remote connection with any organic or functional derangement of these organs, is improved by the use of St. Mary's thistle, nux vomica, or nut-galls, *Rademacher* and his followers consider it as a proof of the dependence of the disease on a primary affection of the organs in question, in spite of the fact that none of these remedies have been proved to have any specific action on these organs whose diseases they are said to cure. The recognition of chronic catarrh with obstruction is facilitated, if there be at the same time a chronic gastric catarrh; but there are cases where the gastric digestion is intact. In the latter cases, the good appetite, the comfort of the patient after eating, and the clean tongue, readily mislead us into seeking the cause of the trouble in other anomalies than in disturbances of digestion. If there be also pain in one or more circumscribed spots in the right hypochondrium, it is often difficult for the physician to make his patient believe that there is chronic intestinal disease. Just at the first flexure of the colon are most frequently found adhesions with the liver, which induce distortions and constrictions, and hence sensitiveness to pressure in this region rather confirms than opposes the diagnosis of chronic intestinal catarrh. The diagnosis of this form of chronic catarrh is materially assisted by the symptoms growing worse if the patient remain constipated for some time. We shall hereafter have frequent occasion to speak of the diagnosis of chronic intestinal catarrh from other abdominal diseases, and will therefore simply again call attention to the fact that it is a very frequent disease, and that in making a diagnosis we should accustom ourselves to first think of ordinary every-day diseases. If this were more commonly done, there would be fewer of those patients who now say that no physician could aid them, and that they did not improve till they began to take *Morrison's* pills.

PROGNOSIS.—The prognosis of intestinal catarrh may be deduced, for the most part, from what we have said of its course. An acute case, causing copious transudation and accelerated movements of the intestines, is usually of not much importance or danger; the diarrhœa may even prove advantageous, by removing irritant substances that have reached the intestines. Moderate intestinal catarrh may also prove beneficial at the period of dentition, in children inclined to hyperæmia of the brain and lungs; but we should disabuse our patients of the belief that all patients *must* have diarrhœa while cutting the teeth, and that at this time we should never try to arrest a diarrhœa. This superstition is widely prevalent and very dangerous; this is why the physician is often not sent for till the child is debilitated and emaciated, and in a very dangerous condition. Under well-timed and suitable treatment even the chronic diarrhœas of children usually offer a favorable prognosis. In accordance with what we have said of its course, even typhlitis and its sequelæ do not often endanger life. The prognosis is worst in the follicular ulcers of the large intestine, particularly when they occur in a person already cachectic, as is usually the case.

TREATMENT.—The causal indications can rarely be fulfilled in intestinal catarrhs depending on congestion, as we can rarely succeed in removing the obstruction to the flow of blood from the intestinal veins. But we can frequently give palliative aid in these cases by attention to the exciting causes; when patients suffer from chronic intestinal catarrh as a result of congestion, we may occasionally, particularly during exacerbations, apply a few leeches at the anus. Sometimes, after repeating this abstraction of blood, at regular intervals, perhaps every four weeks, there may subsequently be periodical loss of blood from the hæmorrhoidal veins, which will greatly relieve the patient. If acute intestinal catarrh has resulted from catching cold, the patient should be put to bed, he should drink a few cups of warm peppermint or camomile-tea, and have the abdomen covered with warm cloths. Patients who suffer from chronic intestinal catarrh, caused by a damp, cold climate, should wear woollen stockings, and change these whenever they have cold feet. Abdominal bandages of flannel, also, are very good in such cases; where women suffer from this disease, they should wear drawers, and in winter these should be made of Canton flannel or some other thick material. (In Greifswald, even the neediest women wear drawers, an article of clothing which elsewhere is only customary among women of the upper classes.) If we neglect this precaution, or if we have too much false modesty, we shall neglect a remedy that is often more important for the welfare of the patient than all other dietetic or medicinal prescriptions.

When chronic intestinal catarrh in children is due to improper nourishment, the causal indications require the regulation of the diet, and attention to this will often be crowned by brilliant success. While the diarrhœa lasts, as has already been explained, the child will rarely stand a milk diet; meat broths suit him best, but still better, finely-shaved raw beef, taken with a little white bread, and a small quantity of good wine, such as pure Tokay or Malaga. Under this treatment, the diarrhœa, which had previously withstood all remedies, often ceases in a short time, and the emaciated child soon recovers its appearance. Concerning the use of calomel, and other customary remedies in gastric and intestinal catarrh, see treatment of gastric catarrh. If the retention of hardened fæces in the colon, or any other part of the intestines, be the cause of the catarrh, the treatment should be commenced with a purgative. If we would treat intestinal catarrh successfully, we should ourselves examine the passages, to see whether there are no hard scybola along with the liquid discharges. The rule of commencing the treatment with a laxative is particularly applicable to the catarrhal inflammation of the colon, which we have designated as catarrhal dysentery. In such cases, one rather large dose of oil often suffices to entirely remove, in a few hours, the abdominal pain, tenesmus, and even the mucous and bloody appearance of the passages. This result is the more striking when the patient has been for days taking mucilaginous soups and opiates, and when, under this treatment, the disease has gradually been growing worse. We can only partly fulfil the causal indications in those cases, also, where twisting and distortion of the intestines, or constrictions, which will be spoken of in the next chapter, lead to habitual constipation, and this again to intestinal catarrh; for, although we can remove the constipation, we cannot get rid of its cause. Such patients only feel well, and can only feel well, while constantly using purgatives, and we have to exercise great care in the choice and composition of the laxatives prescribed. The rule of giving as simple a prescription as possible does not answer in those cases where we wish to prescribe a purgative that will act well for months. Compositions of rhubarb, jalap, aloes, and colocynth answer better than either of these remedies alone; but, as patients often have copious stools, which are, at most, pulpy, not watery, we are often obliged to try for a long while before we find the proper remedy and the suitable dose. Practitioners can obtain at Berlin several packages of *Strahl's* domestic pills, No. II. and III.; then the patient can try for himself how many of each he must take to produce the desired result. By careful attention to keeping the bowels regular, wonderful results are sometimes attained in this disease. Enemata alone, particularly of cold water, rarely answer the purpose, at least for a length of time, but they may be used as adjuvants. The action of laxatives is greatly facilitated by certain dietetic rules, which, however, are not always explicable. Some patients find it advantageous to drink a few glasses of water, or to smoke before breakfast; others to eat bread and butter with their coffee, and most persons are benefited by stewed vegetables, particularly stewed prunes, with their dinner. Regular exercise, on foot or horseback, and other bodily movements, aid the treatment, but we should not over-estimate their value. Finally, we should constantly urge the patient to at least attempt to have a stool at a regular hour every day.

*Bretonneau* and *Trousseau* praise belladonna as the most efficient remedy in habitual constipation, and "dyspepsia accompanied by sluggishness of the large intestine." They give belladonna alone (pulv. belladonnæ, extract belladonnæ, each gr. $\frac{1}{6}$—gr. $\frac{1}{4}$), not, as some physicians do, in combination with drastic purges. Although I cannot agree with all the laudations which *Trousseau* expends on belladonna, not having witnessed its "efficacité merveilleuse" in all patients with habitual constipation, I have, nevertheless, been agreeably surprised by its decided effect in many cases of this affection. Some patients have assured me that, from the time they began to take the belladonna pills, they had felt like new beings, and, particularly with the last prescription, they had less disagreeable sensations than with the former ones. Unfortunately, I cannot at present distinguish the cases of habitual constipation where belladonna is indicated from those where it is not. It is to be hoped that future observations will determine the cases proper for the use of this remedy, which is so excellent in some forms of habitual constipation.

In typhlitis stercoralis, the causal indications also require the removal of the masses collected in the cœcum and ascending colon, it is true, but this is to be done with care. If the case be recent, and unaccompanied by vomiting, we may give a full dose of castor-oil ( ℥ ss—℥ j), but if vomiting has begun, and the oil administered be rejected, we should cease the attempt of giving internal remedies to cause a passage, and, above all, we should not be led into the error of giving drastic purges. As long as there is an insuperable obstacle to the passage downward of the contents of the intestines, all remedies that increase the movements of the bowels cause their contents to move upward, and induce vomiting. In the latter cases, the clysopompe [Davidson's syringe] is an invaluable remedy; it cannot be replaced by a simple syringe, even if we give several injections in succession. We may inject as much as four pints of liquid; as pure water is readily absorbed in the large intestine, we should add to it salt, oil, milk, or honey. Vomiting usually ceases after a moderate evacuation, or even when the passage of a few badly-smelling, crumbly masses shows that the fluid has reached the fecal collections, and has softened and set them in motion. But if there has been a free evacuation, we must not be misled, by the swelling in the cœcal region, into continuing the evacuant treatment. If the intestinal wall or the peritonæum be extensively inflamed, we shall increase the pain and inflammation by continuing to excite the movements of the intestines.

The indications from the disease never require bleeding in acute intestinal catarrh, and even leeching may be dispensed with, except in the treatment of typhlitis. But, in the latter disease, the application of 10—20 leeches in the right iliac region, and the employment of cataplasms, to keep up the bleeding, are usually very beneficial, and the operation should be repeated if the pains recur. As we mentioned when speaking of cholera morbus, cold suits those cases where the hyperæmia is excessive, and is accompanied by moderate transudation into the intestine, as well as in the severe forms of catarrhal enteritis, which occur after extensive burns, and are accompanied by great pain.

The best mode of using cold is by applying cloths, wrung out of cold water, to the abdomen. In the chronic forms of intestinal catarrh, particularly those combined with obstruction, irritating and warming compresses are suitable; among these, *Preissnitz's* compress is extensively used. We let the patient wear a wet towel, covered with a dry one, during the night only, or renew it several times daily. In those cases of chronic intestinal catarrh accompanied by the production of tough mucus, the same mineral waters are indicated as in the analogous form of gastric catarrh. The astringents, also, particularly nitrate of silver and tannin, may, by their astringent action, moderate the relaxation of the mucous membrane, decrease the hyperæmia, and so answer the indications from the disease. Besides these remedies, of which nitrate of silver, in small doses, particularly deserves trial in the chronic catarrh of young children, we may use catechu, kino, colombo, cascarilla, etc.; but the circumstances where any particular one of these remedies deserves the preference are still obscure, and we employ one when we find another unserviceable. Employment of astringents, in the form of enemata, is only advisable when the catarrh affects the large intestine, as even large enemata will not pass the ileo-cœcal valve and enter the small intestine. In follicular ulcers, which particularly occur in the lower part of the large intestine, enemata of nitrate of silver (gr. ij—vj to ℥ vj), sulphate of zinc, or tannin (ʒ ss to ℥ vj), are very useful, and are preferable to all other remedies, but, unfortunately, they are not well borne in all cases.

In cases where the diarrhœa is to be regarded as an injurious, rather than as a favorable symptom, the indications are to arrest it. It is easy to tell, in each case, when the time has come for arresting the discharges. No general rules for this can be given. We usually first attempt to attain our end by dietetic rules, by prescribing mucilaginous drinks, oat-meal, rice, or barley-water, or give soups made of parched meal; and these prescriptions are worth trying. I will not discuss the question as to whether mutton-broth, particularly when fat, will cure diarrhœa, as is popularly believed. Besides mucilaginous drinks, slightly astringent liquids, which are not exactly medicines, are usually prescribed; red wine, infusions of dried whortleberries, roasted acorns, etc. These also may prove serviceable, and are worthy of trial in slight cases. The astringents mentioned above, when speaking of the indications from the disease, may also be named among diarrhœa remedies. As we have already said, nitrate of silver is particularly serviceable in the chronic diarrhœa of children, while catechu in large doses (ʒ ij to ℥ vj of mucilage, a tablespoonful every hour or two) is often surprisingly efficacious in the diarrhœa of adults. In proportion to the advantage from its use, acetate of lead is too dangerous a remedy to merit extensive employment. By far the most certain and useful remedy is opium, little as we positively know of its manner of action. If a diarrhœa seem dangerous, and we wish to check it quickly and certainly, we may use laudanum (℈j—ʒss to ℥vj of mucilage, or weak infusion, of ipecac. Give a tablespoonful every hour). Opium given by enema is not less serviceable than when given by the mouth.

Intestinal disease is only one symptom, and is not even a constant symptom, in typhus fever [Professor N. divides typhus fever into exanthematous or *typhus*, and abdominal or *typhoid* fever. This explains why he does not consider the bowel affection as by any means a constant symptom]; hence it is impossible to fully describe the typhous disease of the intestine without depicting typhus fever. Moreover, typhous bowel affections are the result of the specific infection, which we are compelled to admit in typhus. On this account, when considering the infectious diseases, we shall speak both of typhus and the accompanying intestinal affection.

The case is somewhat different with the intestinal affections in Asiatic cholera and dysentery, induced by miasma; for, in these affections, disease of the intestines is very rarely absent, and all the symptoms of cholera and dysentery may be deduced from the bowel lesions. Hence, we have spoken of cholera morbus and catarrhal dysentery in the present section, and will treat of epidemic cholera and epidemic dysentery in a future one, because we believe that, from a proper interpretation of the etiological causes, the latter must be regarded as infectious diseases.

## CHAPTER II.

### PERFORATING DUODENAL ULCER.

THE cases of perforating ulcer of the duodenum, scattered through the journals, hand-books of practice, and monographs, on the diseases of the duodenum, or of the intestine, were first collected by the industrious and meritorious labor of *Krauss*, and were so carefully analyzed that it is now possible to state something positive about this disease which is probably not very rare.

ETIOLOGY.—From the great resemblance, as well of the anatomical appearances as of the symptoms, course, and results, it is more than probable that perforating duodenal ulcer is due to the same processes that cause perforating ulcer of the stomach; hence, that it is not a proper ulceration, but a necrosis, and a solution of the necrosed part of intestine by the gastric juice.

It is difficult to give any thing definite concerning the frequency of duodenal ulcer, for doubtless some cases that have not led to perforation, and still more, some cicatrices of healed duodenal ulcers, have been overlooked at the autopsy. In one thousand *post-mortem* examinations made at the Prague institute for pathological anatomy, *Willigk* found perforating duodenal ulcer only twice, while in seventy-four cases he found either ulcers or their cicatrices. Perforating duodenal ulcers appear to be more frequent in men than in women; just the opposite of what occurs in ulcer of the stomach. It is hardly ever seen during childhood; most of the cases collected by *Krauss* occurred during middle age. It does not appear, from the analysis of the cases known, whether certain causes, particularly burns of the skin, induce this disease.

ANATOMICAL APPEARANCES.—The most frequent seat of the ulcer is the upper horizontal portion of the duodenum; in some few cases it has been observed in the descending portion, and in one case in the lower horizontal portion (*Krauss*). *Lebert* says that perforating ulcers may occur in any portion of the intestinal canal. I myself have seen an ulcer, with all the characteristics of simple perforating ulcer, in the upper third of the small intestine, in a public officer, aged fifty-six years. In recent cases the edges of the ulcer are sharp and not swollen, the loss of substance in the mucous membrane is more extensive than in the muscular coat, and greater in this than in the serous. Ulcers that have existed some time are surrounded by thickened edges, indurated by newly-formed connective tissue. In some cases the floor of the ulcer is formed by neighboring organs to which the duodenum has become adherent before its complete perforation. The liver, pancreas, gall-bladder, and posterior wall of the abdomen, have been observed as coverings of duodenal ulcers. The progress of the destruction from the duodenum to the adherent gall-bladder occasionally causes a fistulous communication between the two. A continuation of the destruction to the adherent abdominal wall may lead to perforation outwardly. Occasionally this, like the perforating ulcer of the stomach, heals, with great retraction of the cicatricial tissue. It may thus lead to stricture of the duodenum. Finally, obliteration of the ductus choledochus has been observed as a result of cicatricial contraction of a healing duodenal ulcer.

SYMPTOMS AND COURSE.—When speaking of round ulcers of the stomach, we mentioned cases where peritonitis which was rapidly fatal, or severe vomiting of blood, was the first symptom from which that severe and dangerous disease could be diagnosticated. Perforating ulcer of the duodenum appears to remain latent until the fatal termination, more frequently than similar ulcers of the stomach. At the same time it should not be said that the patients have been perfectly well until the appearance of these fatal symptoms; on the contrary, it seems that slight dyspepsia, a feeling of fulness after eating, and sensitiveness to pressure in the upper part of the abdomen, have preceded the perforation or the vomiting of blood for a few days; but these symptoms have remained unnoticed, or have not led to the diagnosis. In another series of cases, the symptoms were as nearly as possible those common to perforating ulcers of the stomach. The cases that have been published do not by any means prove that cardialgia and vomiting occur later in perforating ulcer of the duodenum than in the same disease of the stomach, and only in a few cases did it appear that the pains were seated rather farther to the right side. In the same way the analysis of published cases shows that duodenal ulcer does not induce icterus more frequently, so that this symptom does not aid in the diagnosis between gastric and duodenal ulcers. The rare occurrence of icterus appears to prove that perforating ulcer of the duodenum is not accompanied by extensive catarrh any more frequently than ulcer of the stomach is. If the catarrh did occur, nutrition would be sooner affected from ulcer of the stomach, and obstruction of the gall-ducts with resorption of bile (icterus), from ulcer of the duodenum. The sudden occurrence of peritonitis after slight disturbance of digestion gives us no more certainty, in the diagnosis between a perforating ulcer of the duodenum and one of the stomach, than does the following group of symptoms, viz., feeling of pressure and fulness after eating, sensitiveness in the epigastrium, cardialgia and vomiting. Perforating ulcer of the stomach being far the more frequent, the probabilities are in its favor. Finally, a number of cases are reported, where duodenal ulcers ran their course with periodical attacks of pain, and where, from the pain being in the right hypochondrium, from their occurrence several hours after meals, and the accompanying symptoms of dyspepsia and acidity, and occasionally from decided enlargement of the stomach, the diagnosis of duodenal ulcer could be made with great probability. But even in such cases we cannot always be certain there is not a cancerous or a simple stricture of the pylorus. At the present time, I am treating two patients who, besides having a dull pressure in the right hypochondrium, complain of an insufferable feeling of fulness after eating, also of a belching sometimes of gases without smell or taste, sometimes of sour and rancid substances. One of these patients *never* vomits, the other rarely; but both appear convinced that there must be an obstruction to the exit of food from the stomach, both insist that the food escapes from the stomach more readily when they remain upright for a few hours after eating; and, in spite of their emaciation and debility, they persist in sitting up for several hours after their meals. No tumor can be found in the hypochondrium; the prominence in the epigastrium can be perceived after a large meal. In both patients the disease has lasted several years. I consider it as not improbable that these patients have an ulcer or a cicatrix in the duodenum.

Absence of vomiting speaks against stricture of the pylorus, but I cannot with certainty exclude a constriction of the duodenum or commencement of the jejunum caused by chronic partial peritonitis (see Chapter III.).

If it be difficult to diagnosticate an open ulcer of the stomach from one that has healed and left a contracting cicatrix, it is impossible to distinguish between an open duodenal ulcer and a cicatrix. The peritonitis caused by the perforation of a duodenal ulcer runs the same course as one depending on perforation of an ulcer of the stomach, only it appears to run its course more rapidly, perhaps from the mixture of bile with the contents that escape into the abdomen. For the symptoms resulting from perforation into the gall-bladder, or externally, I refer to the monograph of *Krauss.*

TREATMENT.—Perforating ulcers of the duodenum are to be treated in the same way as perforating ulcers of the stomach; by strict regulation of the diet, the use of alkaline and alkaline-saline mineral waters, particularly the warm springs of Karlsbad and Ems, and under some circumstances by nitrate of bismuth, and nitrate of silver. If there be severe cardialgia, narcotics are indispensable.

## CHAPTER III.

### CONTRACTIONS AND CLOSURES OF THE INTESTINAL CANAL.

THE varied processes which induce constriction or closure of the intestine are best treated of in the same chapter, as the greater part of the symptoms excited by them are common to all.

ETIOLOGY.—1. Contraction or closure of the intestine may result from *compression.* The rectum is most frequently compressed either by a retroverted uterus or a pelvic tumor, such as fibroid of the uterus, ovarian cysts having an unusual location, or by tumors and abscesses starting from the pelvic bones or other tissue. Occasionally an overfilled or cancerous portion of intestine compresses the portions of intestine lying under it; or a piece of mesentery, drawn down by the intestine belonging to it, being in a large hernial sac, compresses portions of intestine lying between it and the spinal column.

2. Constriction of the intestine may be caused by structural changes of the wall of the intestine. The different forms of stricture of the bowel come under this head. Those resulting from cicatrization of intestinal ulcers, particularly the catarrhal, follicular, or dysenteric,

are the most frequent. Cicatrization of tuberculous (scrofulous) ulcers rarely, and of typhoid ulcers never, leads to stricture of the intestine. Cicatricial strictures occur in the rectum also, after the healing of syphilitic ulcers or of wounds. Simple stricture due to hypertrophy of the walls of the intestine is more rare than that which occurs in the œsophagus and pylorus from the same cause. Lastly, we must mention those strictures induced by neoplasia, particularly carcinoma, of which we shall hereafter speak.

3. The intestine may be closed by rotation on its axis; even half a rotation closes its calibre. The closure may result either from a portion of intestine rotating on its own axis, or from the mesentery, or part of it with the intestine attached to it, being twisted on itself, or from a portion of mesentery with its intestine being wound around another loop of intestine. A long and relaxed mesentery predisposes to the occurrence of rotation on the axis; the mechanism of this is obscure.

4. Closure of the intestine may result from internal strangulation, or incarceration. This occurs when a portion of intestine enters any fissure in the abdomen, or gets behind a ligament stretched there, and thus becomes constricted. A portion of intestine may be thus strangulated in the foramen of Winslow, or in a congenital or developed fissure in the omentum or mesentery. The bands which most frequently cause strangulation are those resulting from peritonitis; they occur between the most different organs, but particularly between the uterus and its surroundings. A portion of intestine may be thrown around the omentum which is drawn downward strongly, or around the vermiform process which has become adherent at its point, and may thus be constricted.

5. The intestine may be closed by one portion of intestine entering another portion; this almost always takes place from above downward. This is called *invagination*, or *intussusception*, as it consists of an inversion of the intestine into itself. When this occurs, there are three layers of intestine, one over the other; the outer one is called the sheath, or intussuscipiens; the middle and inner one are called the intussusceptum. The mucous surface of the external and middle layers and the serous coat of the middle and internal layers are brought in apposition. The mesentery lies between the middle and internal layers. As this is attached at its root, it is rendered tense by this inversion, and hence exercises traction where it is inserted into the intestine. In consequence of this one-sided traction, the invaginated portion of intestine is distorted; its opening is drawn from the middle toward the side of the sheath, and it is elongated to a narrow fissure. If there be quantities of intestinal contents pressing downward, the invaginated

portion is constantly driven deeper in, and the inversion becomes more complete. Intussusception is found in both the small and large intestine. The lower end of the small intestine not unfrequently enters the large intestine; and cases have been observed where the ileo-cæcal valve was close to, or even projected out of, the anus. Intussusceptions mostly occur in the course of chronic diarrhœas; it is most probable that they are caused by a portion of intestine contracting strongly, and, by elongating and moving forward at the same time, entering the non-contracted portion just below; part of the latter is drawn along and inverted with the contracted portion. New peristaltic movements force the invaginated portion of intestine farther and farther into the outer portion, until the resistance from the mesentery, or the adhesion of the parts pushed into one another, arrests the progress of the inner portion. Occasionally, particularly in the bodies of children who have died of hydrocephalus, we often find one or more intussusceptions, which are usually short; these have occurred during the death-agony, as is shown by the absence of all signs of inflammation. They also appear to be caused by increased and unequal contraction of the intestines, by which the contracted portions are forced into the larger. It is worthy of remark that increased movements of the intestines, which may even be perceived through the abdominal walls, are seen just before death in animals, even after paralysis of the cerebro-spinal system has occurred.

6. Finally, the intestines may be closed by extensive accumulations of hard and dry fæces, or by stony concretions consisting of hardened fæces, or precipitates of the triple phosphates and lime-salts. This form of closure may be just as complete, and the symptoms durinig lfe may be just as threatening, as in those caused by rotation of the intestine on its axis, by internal strangulation, or by invagination. Cases where fecal vomiting and obstinate constipation were overcome by large doses of metallic mercury and similar remedies are not to be blindly taken as examples of cures of internal strangulated hernia, etc., but rather prove that retained fæces may excite the combination of symptoms, which is usually designated as ileus or miserere. Complete closure of the intestines by masses of fæces occurs most readily at those places where mechanical obstructions constantly oppose and retard the progress of the contents of the intestines; hence it is more likely to take place above the bent portions and the adhesions, of which we spoke in a previous chapter, above compressed portions, or above the various forms of stricture of the intestine. In other cases a sub-paralytic state of the intestinal muscles, or a diminution of the secretion from the intestinal mucous membrane, appears to favor the collection of the obstructing fæces. Lastly, the use of food which forms a great quantity

of hard fæces, such as vegetables, or bread containing much bran, or even of badly-prepared asparagus, may induce this form of obstruction of the intestines.

Anatomical Appearances.—It would take too long to speak in detail of the different tumors that may compress the intestine. The pathological anatomy of strictures of the intestine is entered into when speaking of the different diseases that cause them. The change of position of the intestines that causes closure of the intestines has been previously described.

Above contracted places we usually find the intestine dilated, and, as it is at the same time elongated, it is abnormally curved. The walls of these portions of intestine are usually hypertrophied, or at least thickened; the cavities are filled with gases and fæces. Below the contraction the bowel appears empty and collapsed. Where gas and fæces have rested for a time, the mucous membrane is usually in a state of chronic catarrh, which, from time to time, becomes acute (see previous chapter).

In closure of the intestine, its vessels, and in some cases those of the mesentery also, are compressed; in consequence of which there is great capillary congestion, which induces decided swelling of the wall of the intestine, severe catarrh of the intestinal mucous membrane, transudations and small hæmorrhages in the serous coat. More or less extensive peritonitis usually accompanies these changes. If the pressure and tension of the vessels be not removed, absolute stasis occurs in the capillaries, and, in consequence of this, mortification of the wall of the intestine. In the latter case there may be a perforation, which almost always causes death from peritonitis. In some few cases the intestine becomes adherent to the abdominal wall before perforation, and a fecal fistula or so-called artificial anus results; these diseases belong to the domain of surgery. In intussusception the mortification of the invaginated part, and its passage through the anus, may effect a relative cure, if a firm adhesion between the external and middle layers of the intussusception have previously taken place; but this place usually remains permanently constricted. This is still more apt to be the case if only the lower part of the invaginated intestine slough off while the upper part becomes firmly adherent to the sheath, so that at this point the intestinal wall shall permanently consist of several superimposed layers.

Symptoms and Course.—The most important symptom of constriction of the intestine is difficult and tedious defecation. But, as many persons suffer from sluggish bowels without any mechanical obstacle impeding the progress of the contents of their bowels, it does not appear improper to insert here some remarks on "habitual constipation."

*Henoch*, who, in his clinic of abdominal diseases, treats very exhaustively of habitual constipation, and gives a very lifelike and accurate account of the inconvenience to which it leads, says very truly that "suffering from constipation" is a very relative term. Some persons habitually only have a passage every second or third day, and still feel very well, or feel worse when they have more; on the other hand, others feel sick if they do not have one or two stools daily. The cause of this difference depends partly on the fact that the former form but little fæces, as they eat food containing but little indigestible material, and as they perfectly assimilate the digestible part of the food; while the latter have a quantity of fæces, because their food contains much indigestible material, or because their power of digestion is impaired. But even persons eating similar food and digesting equally well show the same difference in the number of evacuations required to keep them feeling well. It is difficult to give a satisfactory explanation of these symptoms, but in most cases they seem to depend on the fact that in some persons the irritation of the mucous membrane, by the retained fæces and the products of decomposition, leads to intestinal catarrh, while in other less susceptible persons the intestine remains healthy. In the latter cases only small amounts of gases form from the fæces contained in the intestines, the abdomen does not become tense, and the diaphragm is not pressed upward, even when the person has no passage for two or three days or more. In the former cases the mucus covering the walls of the intestine acts on the contents of the bowels as a ferment, and by their rapid decomposition quantities of gas are formed; the abdomen is puffed up, and, even after a short retention of fæces, we have the inconvenience described in a previous chapter. To this description we have to add a few symptoms that depend more directly on collection of fæces in the lower portions of the bowels, particularly in the flexure of the colon and in the rectum. Occasionally patients have an "unsatisfactory feeling," as *Henoch* aptly calls it, after stool; they feel as if there were still masses in the intestines, which should have been passed. This feeling alone gives them great discomfort, and puts them in a disagreeable frame of mind. But, besides this, there are often symptoms resulting from the pressure of the full intestines on the neighboring blood-vessels and nerves. Pressure on the iliac veins rarely causes œdema of the feet; but patients with habitual constipation usually suffer from cold feet, a very annoying symptom, which is most readily explained by the impeded return of the blood from the feet. Dilatation of the vessels in the walls of the rectum most frequently result from pressure on the hypogastric veins, and occasionally there are ruptures of these dilated vessels. The significance of these varicose vessels and the hæmorrhages (blind and bleeding piles) is overvalued by the laity, who usually regard them as the cause and not as the result of their trouble. In the same way the escape of blood from the pudic veins, or, in women, from the uterine veins, may be impeded. In consequence of this, in most women who are habitually constipated, there is hyperæmia of the uterus, which shows itself by very abundant menstruation and uterine catarrh, and which subsequently often leads to important disorders of nutrition of the uterus. Thus we see that the notorious *Morrison's* pills may not incorrectly be said to benefit menstrual difficulties and fluor albus. Men with habitual constipation may have frequent erections and seminal emissions, induced by the impeded escape of blood from the pudic veins; if they were previously worried about their feelings, they are absolutely frightened by this symptom. Lastly, the pressure of the loaded intestines on the sacral plexus may cause neuralgic pains in the legs, or, what is more frequent, a feeling of numbness. The causes of habitual constipation, which does not depend on curvatures and adhesions of the intestines, or on the various forms of constriction, are rather obscure. The disease occurs more frequently in women than in men, and not unfrequently develops in growing children. A slow movement of the intestine appears most frequently to induce it; but there is scarcely any explanation of this sluggishness of the intestinal muscles. The bad habit, of repeatedly restraining the fæces forcibly, induces habitual constipation in some cases. "Sedentary habits," also, such as are common to students and persons of certain occupations, are likewise properly classed among the exciting causes of this affection. Still it is remarkable that perseveringly walking does not, by any means, render defecation as easy as might be expected. Patients with habitual constipation usually become indefatigable walkers, without thereby attaining the goal which is often the object of their whole desire and endeavor (*Henoch*). In some cases, the habitual constipation is due to chronic intestinal catarrh, which, like chronic gastric catarrh, as we have shown, induces a sub-paralytic state of the intestinal muscles, in spite of the thickening of the walls of the intestines that it causes. Hence people who have led a luxurious life often suffer from habitual constipation. We often meet persons who, at the university, were great beer-drinkers, and were most jovial and popular fellows, who, a few years later, have become ill-tempered and peevish, and have no thoughts beyond whether they "will have the longed-for passage to-day." Recently, inactivity of the abdominal muscles has been classed among the causes of habitual constipation, and cases have been described where the patients accustomed the abdominal muscles to exercise, and were thus cured of their constipation. The excessive stretching and relaxation of the abdomen remaining after frequent pregnancies, particularly after twins, appear to me much more injurious than the diminished activity of the abdominal muscles. Such women, upon whose abdomen it seems cruel to press, from a feeling that we should break through the walls, almost always suffer from habitual constipation, and they cannot strain much; and in these same women there is always abdominal plethora and chronic intestinal catarrh, which alone would sufficiently explain the retarded defecation. (We may readily understand that, under these circumstances, a dilatation of the blood-vessels in the abdomen can easily occur; when we consider that, normally, they are subjected not only to the pressure of the atmosphere, but to that caused by the tension of the abdominal walls, and consequently, when the latter are relaxed, they are deprived of one important aid to the preservation of their normal condition.)

As habitual constipation, then, has so many causes, that its presence alone does not justify the diagnosis of stricture of the intestine, the question arises, How shall we recognize such a cause of constipation? The observation of the fæces may aid us here. In the ordinary forms of constipation, sausage-shaped fæces, of extraordinary size, are often passed; in stricture of the intestine, on the contrary, particularly when seated at the lower part of the intestine, they often have a very small calibre, and consist of small rolls, scarcely as thick as the finger, or of small round masses, like sheep's dung. Important as this symptom is for the diagnosis of stricture of the intestine, we must still remember that it may also occur after long starvation, particularly after long-continued disease. The empty intestine, which is tightly contracted at such times, appears to expand only gradually to its former calibre. Even spasmodic contractions of the sphincter occasionally cause this form of fæces. Hence, before we can diagnosticate stricture of the intestines from this symptom, the above states must be excluded. The history may give another aid in diagnosis. We have already explained that cicatricial contraction is the most frequent cause of stricture. Hence, if habitual constipation and a peculiar form of the fæces occur after a long attack of dysentery, or after diarrhœas which seemed to depend on ulcers of the intestine, the presumption is in favor of stricture. In the same way, in other cases, we may suspect, from the history, that there is a retroversion of the uterus, or some kind of tumor in the pelvis, which is compressing the intestine. In forming a diagnosis, we should also avail ourselves of physical examination. Prominence of the abdomen at any part, and a remarkably full percussion-sound at this point, if found at repeated examinations, show that part of the intestine, above a constricted portion, is dilated. Finally, when we suspect stricture of the intestine, we should never neglect to examine the rectum with the finger. If we can reach no stricture with the finger, we should use an elastic catheter. We may be deceived by the catheter striking against the promontory of the sacrum, or by a fold in the wall of the intestine, preventing the further entrance of the instrument. Spasmodic contractions of the rectum may also deceive unaccustomed observers. In women it is just as important to make a vaginal examination, to satisfy ourselves about the position, size, and form of the uterus, and find if there be any tumors in the pelvis.

The symptoms of closure of the intestines, which subsequently become terrible, and very dangerous, are often slight, and apparently free from danger at the outset. The patients feel puffed up, have colic, periodically appearing and disappearing, think they have made some error in diet, and take some camomile-tea, or a slight laxative. The physician is not usually called till these remedies fail, and, in spite of them, the pains increase, and the bowels do not move, or when nausea and vomiting occur. A careful and experienced physician always considers this combination of symptoms as disagreeable and threatening. The first thing to do is carefully, without over-modesty or forbearance, to examine those parts of the body where hernia may occur. Woe to him who trusts that the patient will, unquestioned, tell him he has a rupture, or who rests contented with his simple denial of the question! The rectum and vagina should be explored just as carefully as the inguinal regions, to find if there be any obstructions to the evacuation of the bowels. In spite of the anxiety that the case causes him, the physician, of course, hopes, at this time, that the symptoms are excited by a retention of fæces, somewhat obstinate, perhaps, but one which may, nevertheless, be overcome. He prescribes enemata, and large doses of castor-oil, with the addition of a little croton-oil. After a few hours he returns, uncalled for, to the bedside of the patient, for the purpose of satisfying his own anxiety. Meanwhile, the enemata have occasionally brought away a little fæces from the lower part of the intestine, but, in most cases, they have had no effect, or it has been impossible to give the patient an enema. In spite of the addition of croton-oil, the castor-oil has remained ineffectual; after taking it, the patient has had great pain, and frequently vomited green masses. At the same time, his appearance has changed; his face is distorted and pale, the skin, particularly on the hands, is cool, the pulse small. Now the sorrowful conclusion becomes more and more certain, that the intestine is closed, and, perhaps, is so obstructed that medical aid can give no relief. The abdomen gradually becomes more prominent; there are severe, straining, bearing-down pains, which the patient calls cramps. These paroxysms of pain are usually followed by nausea, and, to the terror of the patient and those around him, the vomited masses constantly become browner and more discolored, and the smell more distinctly feculent. There has been much dispute as to whether stercoraceous vomiting could result from obstruction of the small intestine, or if it could only occur in closure of the large intestine, where the formation of the fæces proper begins. We should bear in mind that even the contents of the ileum, particularly if they have been there long, may have a feculent odor, and that in so-called fecal vomiting actual fæces are rarely, if ever, vomited. I consider it improbable that the contents of the large intestine should pass the ileo-cœcal valve, and enter the small intestine and stomach. There are various views, even, concerning the origin of retrograde movements of the contents of the intestine. *Betz* not only denies all influence of the contractions of the intestines on the backward movement of the contents, but he even believes that they rather impede than aid the normal progress of the contents from the stomach toward the rectum. In the act of vomiting, abdominal pressure unmistakably plays the chief part; it is this, aided by the above-mentioned contraction of the pyloric portion of the stomach, which evacuates its contents. The contents of the intestines appear to enter the stomach, because, during the contraction of the intestines, the masses cannot pass downward, and are driven upward. We will not discuss the question as to whether this occurs regularly, or whether the contraction of one portion of intestine immediately follows the contraction of the portion just below it—that is, whether there be an actual antiperistaltic motion. At all events, it is evident that, as long as the obstruction exists, all drastics, by increasing the contraction of the intestines, must induce vomiting. In some cases the disease runs along for eight to fourteen days, or longer, with these symptoms, which may even temporarily remit. During the attacks of pain which usually precede the vomiting, according to *Watson's* graphic description, we may feel or see in the abdomen "immense coils of intestine, as big, perhaps, as one's arm, rise and roll over, like some huge snake, with loud roarings and flatulence. The distended bowel strives with all its power, but strives in vain, to overcome the opposing barrier." The patient, now fearfully disfigured, has a ghostly look, a dirty color, his face is covered with cold sweat, his hands cool, his pulse imperceptible, while the mind remains long unclouded; finally, he dies exhausted, with the symptoms of general paralysis.

The picture is somewhat different when extensive peritonitis occurs early in the obstruction. Then the abdomen is puffed up much sooner; it becomes excessively tense, and so painful that even the slightest pressure is not borne. As the exudation occurs almost exclusively between the inflated intestines, it can rarely be recognized by the dull percussion-sound at the dependent parts of the abdomen. The patients do not toss around on the bed, like those above described, but lie still on their back, carefully avoiding all movement, because it increases their pain. The pulse is very frequent, the temperature very high; the diaphragm, pressed upward, compresses the lungs, and the respiration is hastened; the obstructed flow of blood from the right side of the heart gives the patient a cyanotic look. In comparison with the symptoms above mentioned, which even now continue, those of peritonitis are so prominent that the former are not sufficiently noticed, and, while the peritonitis is recognized, the internal strangulation, or other obstruction of the intestine causing it, escapes observation. We should make it a rule to consider "rheumatic peritonitis" a very rare disease, and, when peritonitis occurs in a non-puerperal patient, to think of perforation, particularly of perforation of the stomach by an ulcer, or of acute obstruction of the intestine. If the disease has occurred quite suddenly, and is not accompanied by vomiting, the chances are in favor of perforation. If it has come on gradually, and there was vomiting at the very first, which continues obstinately, or if fecal vomiting occurs, there is, most probably, obstruction of the intestine. In the latter case, the course is usually much more rapid; even after a few days, there is usually great collapse, general paralysis, and almost always a fatal termination.

During life it can hardly be said, in any case, whether the group of symptoms that we have described depends on a rotation, internal strangulation, intussusception, or an obstruction of the intestine by hardened fæces or stony concretions. We have the least certainty in diagnosticating rotation of the intestine on its axis. The suspicion of internal strangulation is somewhat supported, if the patient has had a previous attack of peritonitis, as the bands, which are the most frequent cause of the strangulation, are almost always remains of former peritonitis. Intussusception occasionally shows peculiarities of symptoms by which it may readily be distinguished from other forms of obstruction of the intestine. Among these is a sausage-shaped tumor, usually of only moderate resistance, which may sometimes be felt in the abdomen, particularly when the walls are not very tense. This tumor cannot be moved much; it is painful, and, on percussion, gives a sound not quite dull. In intussusception, moreover, the calibre of the intestine is often not closed so completely as in other forms, so that, occasionally, small quantities of fæces, or intestinal gases, are evacuated. Moreover, from the compression of the veins of the mesentery, which is likewise invaginated, there is great congestion of the invaginated portion of intestine, which may readily induce rupture of vessels in the mucous membrane, and bloody or bloody-mucous passages. This symptom is peculiarly important in the diagnosis of intussusception in small children, in whom the disease occurs, proportionately, very often. (In these cases the cœcum and ascending colon are almost always inverted into the lower part of the large intestine, and into the rectum; the constipation is not always absolute; vomiting rarely becomes fecal, and the puffing up of the abdomen is usually moderate as peritonitis is rare. On the other hand, the bloody or bloody-mucous dejections are rarely absent.) The diagnosis of intussusception is beyond doubt, when, from the rectum, we can feel the slit-shaped mouth of the invaginated portion of intestine, which is usually turned toward the wall of the rectum, or, if this sloughs off, and is evacuated, somewhat mortified, but still recognizable. Obstruction of the bowels by hard fecal masses is readily recognized when these can be felt in the rectum. In other cases, a hard movable tumor in the abdomen leaves little doubt that it is formed of hard fecal masses or stony concretions, and that these obstruct the intestines. If the patient has previously suffered from the symptoms of constriction of the intestines, and if these have suddenly increased to those of absolute obstruction, according to what was above said, it is probable that the sudden obstruction has been induced by hard masses of fæces. Above all, the favorable course of the disease, the sudden disappearance of the symptoms after the passages of a quantity of fæces, speaks in favor of the latter variety of obstruction.

The diagnosis and prognosis of obstruction of the intestine are evident from what we have said of the symptoms and course of the disease.

Treatment.—The treatment of habitual constipation has been as fully discussed as the plan of this work allows, when speaking of the causal indications in the previous chapter; a discussion of the advantages of and objections to individual laxatives does not come within its scope. I have no personal experience of the result of the Swedish movement-cure in habitual constipation. Electricity, which has also been recommended, comes under the head of gymnastics; contractions of the abdominal muscles may be caused by applying the electrodes to the skin of the abdomen, and they may be strengthened by repeated applications. Application of the electrodes to the abdomen has no effect on the movements of the intestines themselves. The proposal to place one electrode in the mouth, the other in the anus, must be regarded as very naïve.

If stricture of the intestine be located in the rectum, the treatment consists in the removal of tumors, or dilatation of strictures, and, where these procedures will not answer, in the formation of an artificial anus; hence it belongs to surgery. Contractions of the intestine higher up can never be radically cured. We have to limit ourselves to placing the patient on a diet which will leave as little fæces as possible. The more threatening the symptoms of the constriction, the more necessary it becomes that the patient should live on eggs, strong broths, and pure muscular meat, with delicate fibre. At the same time, we must secure regular evacuations by enemata and laxatives.

Those cases of obstruction of the intestine that are caused by hardened fæces and stony concretions are far more amenable to treatment than those caused by changed position of the intestines. This is particularly true of the obstructions of the rectum by fæces, which are often collected in astonishing masses above the sphincter. A prudish physician, who does not venture to ask for a local examination, will prescribe laxative after laxative for days, in such cases, without any benefit; while a physician who has no false modesty, and takes no refusal when it is a question of knowing the disease more thoroughly, obtains wonderful results. It often requires great pains and untiring patience to make a passage with the finger, the handle of a spoon, or corn-tongs, for enemata which, at first, would not enter, and to pass an elastic tube through these masses, and give softening enemata. The task becomes more difficult if the hardened fæces be higher up. We may here refer to what we said of the removal of impacted fæces when speaking of the treatment of typhlitis stercoralis. At first we attempt to induce a passage by a few spoonfuls of castor-oil, or by large doses of calomel; to each dose of the former we may add half a drop of croton-oil. If these remedies prove ineffectual, and increase the vomiting, we should confine ourselves to the use of the clysopompe [Davison's syringe], which certainly offers the most chance of softening the hard masses which are usually in the large intestine. We must not be discouraged if the first injection does not produce an effect, but must repeat it two or three times daily. In one case that I have seen, it was not till after four days' assiduous use of the pump that small, friable, greatly-discolored masses of fæces, which had a horrible odor, were mixed with the fluid injected. And not till the following day were there large quantities of similar appearance. In desperate cases we may use pure quicksilver; from a few ounces to a pound or more of this article may be swallowed. It cannot be denied that, in some cases, where all other remedies failed, the weight of the mercury broke through the obstruction. Rotations and internal strangulations can rarely be diagnosticated with sufficient certainty to justify gastrotomy, which, to be successful, should be performed as early as possible. It is not impossible that quicksilver may, by its weight, remove a rotation of the intestine, or cause the reposition of a strangulated part by the traction that it exercises on it before reaching it; however, there is some doubt about the diagnosis of the cases where this result is said to have been attained. Since, in intussusception, the inversion of the intestine almost invariably takes place from above downward, there is a contraindication to the exhibition of laxatives, which would force the invaginated portion still deeper into the sheath. This is still more true of quicksilver. When the intussusception has been recognized early, we may perform gastrotomy, as has been successfully done in some cases. If we can reach the invaginated intestine through the rectum, we should attempt to replace it by carefully introducing an œsophageal bougie that has a sponge fastened to its end. This procedure has been particularly successful in some cases in children. If we cannot reach the invaginated part, we may inject large quantities of liquid, or blow air into the rectum with an air-bag, so as to press back the invaginated portion, if possible. As soon as severe peritonitis has occurred, these procedures can be of no use, but may do harm, as the portions of intestine have become glued together. Then we should confine ourselves to large doses of opium, and to covering the abdomen with cold compresses. The same treatment should be followed where the symptoms of extensive peritonitis occur with other forms of obstruction of the intestine.

## CHAPTER IV.

### SCROFULOUS AND TUBERCULOUS DISEASES OF THE INTESTINES AND MESENTERIC GLANDS.

Etiology.—Tuberculosis of the intestine and mesenteric glands is not, by any means, so frequent as is taught; many so-called tuberculous diseases of these parts are not at all due to the formation of miliary tubercle, the only form of tubercle that we recognize, but to a cheesy degeneration of the intestinal follicles and mesenteric glands.

The solitary glands, and glands of *Peyer* of the intestine, which are known not to be secretory organs, but elementary lymphatics, are sympathetically affected in the different diseases of the intestinal mucous membrane. In acute and chronic catarrhs of the intestine they are always found more or less swelled, and projecting above the surrounding parts. This swelling of the follicle, which depends partly on the increase of cellular elements, and still more on increased absorption of fluid, usually disappears without leaving a trace, on the subsidence of the catarrh. But, under some circumstances, it becomes more decided and obstinate; the cellular hyperplasia particularly attains a high grade, and then, as occurs elsewhere, when there is an extensive collection of cellular elements, atrophy, or an incomplete metamorphosis into fat (cheesy degeneration), readily occurs. The mesenteric glands that derive their lymph from the intestinal mucous membrane usually participate in its diseases, just as the small lymphatics located in the walls of the intestine itself do. In acute and chronic catarrh of the intestine and cellular hyperplasia, the increased absorption of fluid causes them also to swell, but this swelling is usually slight and temporary; but, under some circumstances, it becomes more decided and obstinate, and as, in such cases, there is also a large collection of cellular elements in the mesenteric glands, they readily undergo cheesy degeneration.

The caseously degenerated intestinal follicles deliquesce after a time, and form small abscesses, filled with cheesy pus, in the walls of the intestine; when the covering of these is perforated small ulcers are left, which may subsequently enlarge by the continuation of the cell-production and the disintegration.

In the caseously degenerated mesenteric glands, deliquescence and perforation of the capsule of the gland and of the peritonæum are more rare; but, on the other hand, condensation and final transformation of the cheesy mass to a chalky pulp, or to a hard chalky concrement, are very frequent.

If we more attentively regard the persons in whom decided and obstinate swelling and cheesy degeneration of the intestinal follicles and mesenteric glands particularly occur, we find that the larger part of them are children, and especially those children in whom the peripheral lymphatic glands and the bronchial glands incline to swell and undergo cheesy degeneration during the course of moist exanthemata, otorrhœa, bronchial catarrh, etc., and which are usually called scrofulous. This circumstance and the entire correspondence of the pathological changes render it consistent to designate this swelling and degeneration of the intestinal follicles and mesenteric glands and intestinal ulcers, consequent upon the former, as *scrofulous diseases of the intestines and mesenteric glands.*

The wide-spread error, that scrofulous ulcers of the intestines are due to tuberculosis, is easily explained: firstly, the caseously infiltrated solitary glands have the greatest similarity to cheesy miliary tubercles; and, secondly, on *post-mortem* examinations, we often find miliary tubercles in the portion of peritonæum corresponding to the ulcers of the intestine. But, if we examine these free from prejudice, and then ask ourselves when the ulcers and when the tubercles probably occurred, we shall often come to the conclusion that the ulcers have existed for a long while, perhaps for years, while the tubercles have been deposited shortly before death. Hence it is just the same in the intestines as in the lungs, where tubercles are finally added to cheesy infiltrations and cavities.

As we have previously explained at length, in some persons the excessive susceptibility and the tendency of the tissues to become the seat of extensive production of indifferent cells, on inflammatory irritation, last beyond the age of childhood. But, as in grown persons different organs are apt to be diseased from those affected in children, the peculiar forms of the disease vary with advancing age. Even at the commencement of puberty, the intestinal catarrhs which were previously so frequent give way to affections of the lungs, etc. Hence, in many autopsies we find calcareous mesenteric glands and cicatrizing intestinal ulcers, while in the lungs there are fresh cheesy masses and progressing destruction. But, very remarkably and inexplicably, experience shows that adults, who are rarely affected by these diseases of the intestinal follicles and mesenteric glands, are often afflicted by them when analogous diseases attack or have already affected their lungs. We may express this bit of experience in another way, by saying: caseous degeneration of the intestinal follicles and mesenteric glands is rare as a primary and idiopathic disease in adults, but occurs quite frequently secondarily, and as a complication of consumption of the lungs.

Except as a secondary tuberculous eruption in the vicinity of scrofulous ulcers of the intestines, tuberculosis of the intestines and mesenteric glands is rare. This is still more true of *tuberculosis of the intestines*, in the strict sense. It appears in many cases as if the secondary tuberculosis of the intestines occurred simultaneously with secondary tuberculosis of the lungs, and as if the proper tuberculous consumption of the intestine affected particularly those who had also the tuberculous form of consumption of the lungs.

Anatomical Appearances.—Caseously infiltrated intestinal follicles and the ulcers resulting from their breaking down are most frequently found in the ileum. Not unfrequently the disease extends thence to the colon, and remarkably often to the processus vermiformis. In some cases the colon alone is affected, while the ileum remains free. These appearances are rarely found in the jejunum, and very seldom in the duodenum or stomach. The number of follicles affected varies. Occasionally a considerable extent of intestine is regularly covered with them; far more frequently the disease is limited to several small spots at some distance apart. In the earliest stage the swollen follicles form slightly-prominent, rather hard gray nodules, about the size of a millet-seed. These become yellow and less hard as the cheesy metamorphosis commences. If the mucous covering have been perforated by the pus, we find round "crater-shaped" ulcers (*Rokitansky's* primitive tuberculous ulcer). In advanced stages the mucous membrane and submucous tissue in the vicinity of the primitive ulcer are the seat of a gray and subsequently of a yellow infiltration. Extensive losses of substance result from the breaking down of the cheesy infiltration and of the infiltrated tissue itself, and the union of several primitive ulcers (*Rokitansky's* secondary tuberculous ulcer). The ulcer extends particularly in the circumference of the intestine, so that finally the ulcers form bands of various width around the interior of the bowel. Infiltration and destruction sometimes go on in the floor of the ulcer, so that it extends in depth also, and may finally perforate the wall of the intestine. As the destruction advances toward the serous coat, a circumscribed peritonitis occurs at the part about to be attacked. Even on external examination of the intestine we can usually tell the points where the ulcers are located, as the serous coat is there cloudy and thickened, and occasionally covered with scanty fibrinous exudations, or attached by these to neighboring loops of intestines. These adhesions of portions of intestine to each other not unfrequently prevent the escape of the contents of the intestine into the abdominal cavity when perforation of the intestine takes place. In such cases the contents do not gush out through the perforation till we break up the adhesions at the autopsy. If capillary hæmorrhage have occurred from the extension of the ulcer, we find the edges and base of the ulcer suffused with blood and covered with dark clots. Complete cure of these ulcers of the intestine is rarely seen. On the other hand, we often find undoubted signs of incomplete cicatrization as a callous, darkly-pigmented or non-pigmented connective tissue forms the floor of the ulcer, and by its retraction appears to have approximated the edges. If the edges of the ulcer approach each other so nearly as to come in contact, they unite together. In such cases a ridge-shaped resistant swelling always remains on the inner surface, while there is a cicatricial retraction on the outer surface of the intestine.

The swelling of the mesenteric glands may be so decided that the individual glands will attain the size of a pigeon's egg, and a collection of them may form a tumor as large as the fist. As long as the increase in size depends on simple cellular hyperplasia, a section will show the glands to be succulent and of a grayish-red color. We often find only a few points of the swollen glands changed to a yellow cheesy mass; in other cases, one or more glands are caseously degenerated throughout. On *post-mortem* examination we frequently find chalky, irregular, sometimes branched, concretions, surrounded by normal or atrophied parenchyma in these mesenteric glands, as a result of cheesy degeneration which may have run its course years before.

In genuine tuberculosis of the intestinal mucous membrane, small gray nodules, either discrete or united into groups, appear in the early stages. If we find such groups of miliary bodies at parts where there are no *Peyer's* glands, it gives the best means of making the very difficult diagnosis between miliary tubercles and swelled follicles. Tuberculous ulcers result from the cheesy degeneration, softening, and breaking down of miliary tubercle; these never become so extensive as the ulcerations dependent on caseous degeneration of the follicles, and in their vicinity we find fresh tuberculous granulations, instead of cheesy infiltration of the tissue.

In the numerous cases of secondary tuberculosis of the peritonæum, we find those portions corresponding to the intestinal ulcer thickened by proliferation of connective tissue, and covered with numerous small nodules. The eruption of tubercles has often spread from these points along the lymphatics to the mesentery.

SYMPTOMS AND COURSE.—It is generally difficult to decide whether a scrofulous child has simple intestinal catarrh, or if there be cheesy degeneration of the intestinal follicles and ulceration of the intestine. The case is suspicious when the passages are preceded by pain, when the abdomen is sensitive to pressure, and particularly when these symptoms are accompanied by a lingering fever. Not unfrequently the diarrhœa disappears for a time, although the intestinal ulcers may remain; the child appears to improve; but some slight error in diet, catching cold, or some other undiscoverable cause, again induces frequent, copious, fluid evacuations. If these renewals of the intestinal affection be accompanied by an increase of the fever, the child soon loses again what strength and flesh he had gained during the interval. Occasionally this variation from good to bad continues for years, and, even when the diarrhœa has ceased for months, we are not at all certain that the ulcers have healed. On autopsy we often find the mucous membrane of the small and even of the large intestine covered with numerous ulcers, when, perhaps, there has been constipation instead of diarrhœa. This is not strange, when we remember that the thinness of the dejections depends solely on the catarrh accompanying the intestinal ulcers, and that the severity of the catarrh varies just as much as the hyperæmia and œdema in the vicinity of a cutaneous ulcer. When the large intestine is free from ulcers and consequently from catarrh, the fluid contents of the intestines entering them become of normal consistence, so that consistent stools are passed during life, and on autopsy we find the lower part of the intestine filled with firm fæces. The longer the disease lasts, the more nutrition is affected by it. The patients are often considered much younger than they really are. Young men of twenty look like boys; girls attain the age of nineteen or twenty without the breasts developing or the menses appearing. Frequently, we do not discover the cause of this retarded development, till, on careful examination, we find that for years they have had repeated attacks of diarrhœa accompanied by fever. Death as a result of scrofulous ulcers of the intestine is far rarer than we might suppose. It most frequently depends on a subsequent consumption of the lungs, or a secondary tuberculosis.

If obstinate diarrhœa join itself to the symptoms of consumption of the lungs, or if it occur as the chest symptoms begin, it is very probable that the intestinal follicles are caseously degenerated, and that ulcers have developed. Even in such cases the diagnosis is not certain, for so-called colliquative diarrhœa occurs during disease of the kidney and consumption of the lungs, without our being able to find any evident structural changes of the intestine on autopsy. Perhaps these diarrhœas are the analogues of the abundant sweatings of the phthisis patient, and of the œdema of the subcutaneous connective tissue; and it is not improbable that thinning of the serum of the blood, a so-called "dropsical crasis," favors the occurrence of serous transudations into the intestines. If the diarrhœa cease, and be replaced by constipation, and great sensitiveness of the abdomen to pressure, there is still greater probability that the previous diarrhœa was caused by intestinal ulcers, for from the above symptoms we may decide that there is peritonitis, and we know that this very frequently accompanies ulcers, which are advancing toward the serous coat.

Caseous degeneration limited to the mesenteric glands, whose remains are often found on autopsy, can hardly be recognized with certainty during life. The intestinal catarrh may long since have disappeared, while the swelling and change of the mesenteric glands continue, just as the swelling of the peripheral glands in many cases outlasts the exanthemata that have caused it. It is very rare for convolutions of glands, even when considerably swollen, to become evident to the touch. We may always suspect this disease when we find a person who has had obstinate diarrhœa, and scrofulous swelling of the peripheral lymphatic glands, with a protuberant belly. In scrofulous catarrh, as we designate intestinal catarrh, which is accompanied by cheesy degeneration and swelling of the mesenteric glands, the nutrition and development of the patient are also affected; the so-called tabes mesenterica does not appear to be due to the impermeability of the mesenteric glands, but solely to the intestinal catarrh. If this be removed, the patients may recover perfectly, and on autopsies we often find chalky masses embedded in the mesenteric glands of robust individuals who have died of acute disease.

We should suspect proper tuberculous ulcers when diarrhœa occurs during decided tuberculosis of the lungs. The secondary eruption of tubercles on the covering of the intestines has no symptoms, except the partial peritonitis which usually accompanies it.

TREATMENT.—In the treatment of scrofulous diseases of the intestinal follicles and mesenteric glands, we should first of all combat the morbid predisposition which excites and maintains them. On this point we may refer to what we have said concerning the prophylaxis and causal treatment of pulmonary consumption, and will only call attention to the important rule, so often neglected, that the patient should be kept in the fresh air as much as possible. When speaking of the treatment of scrofula, we shall treat particularly of the indications for cod-liver oil, acorn-coffee, walnut-leaf-tea, as well as of the use of alkaline springs. Cod-liver oil does not by any means increase the diarrhœa in all cases, so that, when its use is indicated, we may try if it will be borne. In other respects the treatment of scrofulous and intestinal ulcers corresponds with that of chronic intestinal catarrh. If the diarrhœa become exhausting, opium will be indispensable, but, before employing this remedy, we should try the astringents and bitters recommended for the treatment of catarrhal diarrhœa. If the abdomen become sensitive to pressure, we may use warm poultices. If the pains increase greatly, we may apply a few leeches to the painful part.

## CHAPTER V.

### CARCINOMA OF THE INTESTINES.

ETIOLOGY.—Cancer of the intestines is far rarer than that of the stomach; it is almost always primary, and is even generally isolated; it is only in solitary cases that the cancer advances from neighboring organs to the intestine. The etiology is perfectly obscure.

ANATOMICAL APPEARANCES.—Cancer of the intestines affects the large intestines almost exclusively, and particularly the sigmoid flexure and the rectum. Only in rare cases do we find numerous cancerous nodules affecting both the large and small intestine; in the latter case they correspond to *Peyer's* glands.

As in the stomach, so in the intestine, we have scirrhus, medullary and alveolar or colloid cancer. We also find the same combinations of different forms of cancer; the degeneration often begins in the submucous connective tissue as scirrhus, and, after it has perforated the mucous membrane, medullary masses arise from the scirrhus base. Cancer of the intestine has a great inclination to spread in the transverse direction, and so form ring-like strictures. The diseased portion of intestine often sinks down in the abdomen from its weight; at first it remains movable, but subsequently usually becomes fixed by adhesions between it and neighboring organs, caused by partial peritonitis, or by the cancer spreading from the intestine to neighboring organs. The development of the tumor may contract the calibre of the intestine to the size of a quill; the stricture is not usually over a few inches in length. Above the stricture the intestine is often enormously dilated and filled with fæces and gas, its walls are hypertrophied, and the mucous membrane is inflamed in various degrees; below the stricture the intestine is empty and collapsed. As we have stated, when speaking of cancer of the œsophagus and pylorus, the stricture may be enlarged by the breaking down of the cancer. Occasionally the destruction of the cancer extends to the peritonæum; when this has been destroyed, the contents of the intestine enter the abdomen, or, if there have been previous adhesions, the destruction attacks neighboring organs. In the latter case, there may be abnormal communications between different portions of intestine, or, if the affected portion of intestine have become adherent to the abdominal wall, there may be a fecal fistula; perforation of the vagina or bladder may be caused by the breaking down of cancer of the rectum. Ulceration of the inflamed part of intestine above the stricture may also cause perforation, and permit the escape of the contents into the abdomen, or lead to abnormal communications.

Symptoms and Course.—In many cases it is impossible to recognize cancer of the intestines with certainty. Patients in whom it develops complain of dull pain, sometimes continuous, sometimes occurring at intervals at a circumscribed part of the abdomen. Besides this, there is habitual constipation, which usually begins before the stricture exists, and is then due to the degeneration of the muscular coat and the interruption of the movements of the intestines at that part. From time to time the constipation becomes peculiarly obstinate; the pain increases, the belly is puffed up, and nausea, vomiting, and other symptoms of obstruction of the intestines occur. If constipation be relieved, the patient feels pretty well again. These attacks recur at shorter intervals, increase in severity, and threaten life more and more. Finally, the constipation cannot be relieved, and the patient dies with the symptoms of ileus. If, up to that time, the appearance and nutrition of the patient had not suffered, if there were no tumor to be felt in the abdomen, and the form of the fæces gave no clew for diagnosis, the disease would be very obscure. It might be known that there was a gradually increasing obstacle to the progress of the contents of the intestines, but the nature of this obstruction would not be certainly known till revealed by autopsy.

In other cases the patients do not die so soon from an acute attack of obstruction of the bowels, but, besides the gradually increasing constipation and the dull pain in the abdomen, the signs of a severe cachexia appear; there are a rapid loss of strength, great emaciation, and a dirty complexion. These symptoms give a presumption for the cancerous nature of the obstruction. If the emaciation increases, and an uneven, nodular, hard, painful tumor, which is at first movable, can be felt deep in the abdomen, through the thin abdominal walls, there will no longer be any doubt about the diagnosis.

If, as is frequently the case, the cancer be in the rectum, or even a few inches above it, the patients complain of severe pain about the sacrum, extending to the back and thighs. The significance of these sacral and spinal pains is often undervalued for a long while, and they are regarded as symptoms of a disease free from danger, particularly when there is at the same time varicose dilatation of the hæmorrhoidal veins, and a passage of bloody mucus from the intestine. But, gradually, the increasing constipation and the peculiar appearance of the fæces become suspicious. The latter have a very small diameter, are sometimes round, again flat and ribbon-like, or are small balls, like sheep's dung. These passages are at first mucous and glairy, subsequently they are covered with blood and pus, and they are evacuated with constantly-increasing pain, which finally becomes excessive. Occasionally the passages occur more readily, after the cancer breaks down, or, instead of constipation, there may be diarrhœa, which cannot be checked. At the same time there are often abundant hæmorrhages, and during the intervals between defecation a discolored, stinking fluid flows from the rectum, corroding the anus and its vicinity. If the wall of the rectum be perforated, and the ulceration advance to the vagina and bladder, there is a most fearful destruction and a most miserable condition. The description of this and the directions for examining the rectum with the finger and the speculum, which give the most reliable information, we leave to works on surgery.

With few exceptions, of which we have already spoken, the course of cancer of the intestine is rather tedious; it always terminates in death. The latter sometimes occurs with the symptoms of ileus, which appear gradually or suddenly; sometimes with the symptoms of excessive marasmus, which is occasionally accompanied, at the last, by dropsy and thrombus of the veins; sometimes death is hastened by peritonitis, which may occur with or without perforation of the intestine.

TREATMENT.—The treatment of cancer of the intestine can only be palliative. We must try to regulate the diet, so that as little fæces as possible shall be formed; it is best to nourish the patient with concentrated broths, soft-boiled eggs, and milk. We should most strenuously insist on a daily evacuation of the bowels, and, for this purpose, should prescribe laxatives which act certainly, and with as little irritation as possible. Castor-oil seems to suit best, and, according to *Henoch's* observation, after it has been used a long time, the disgust which most patients have for it subsides. For other points we refer to the treatment of stricture of the intestine as described in Chapter II.; and for operative procedures, to works on surgery.

## CHAPTER VI.

### INFLAMMATION OF THE CONNECTIVE TISSUE IN THE VICINITY OF THE INTESTINES; PERITYPHLITIS AND PERIPROCTITIS.

By perityphlitis we understand the inflammation of the connective tissue which attaches the ascending colon to the iliac fascia. In far the greater number of cases this inflammation is propagated from the cœcum and ascending colon; in other cases it is an independent disease; it is then usually called rheumatic perityphlitis; lastly, it occurs late in typhus, septicæmia, puerperal fevers, etc., and then belongs to the so-called metastatic inflammations. The exudation deposited may be absorbed, and the disease end in recovery; but more frequently the inflammation leads to diffuse necrosis of the inflamed connective tissue, and large abscesses form which may extend upward to the kidneys, and downward even below Poupart's ligament, to the inner part of the thigh. Lastly, the posterior wall of the cœcum and of the ascending colon, the anterior wall of the abdomen or the skin of the thigh, may be perforated; or the contents of the abscess may escape into the abdomen and cause peritonitis.

If the disease develops from a typhlitis, after the superficial tumor due to the inflamed cœcum has disappeared, there remains a painful tumor lying farther back. This is covered by the inflated cœcum, and hence gives a clear percussion-sound. Frem the pressure of the tumor on the nerve-trunks, there is often pain, or a dull feeling in the corresponding leg, and from the pressure on the veins there is œdema. If there be resolution of the inflammation, the tumor becomes smaller, the pain less, and the patient quickly recovers. If it leads to suppuration and formation of abscesses, the tumor increases; in favorable cases, fluctuation appears sooner or later in the abdomen or thigh; when the abscess opens, purulent masses, mixed with mortified connective tissue, are evacuated, and if the strength of the patient hold out, cure may result in these cases also; in other cases death results from exhaustion. If the contents of the abscess escape into the ascending colon after perforation of its posterior wall, the result is usually favorable; but if, on the contrary, they break through into the abdominal cavity, the resulting peritonitis soon causes death. The course of rheumatic perityphlitis is perfectly similar, while in the metastatic form death usually results from the constitutional disease before suppuration and perforation take place.

At the commencement of the disease, as in typhlitis, we apply leeches; this application may be repeated several times; subsequently warm poultices may be used. The abscess should be opened as soon as there is fluctuation.

*Periproctitis* is an inflammation of the connective tissue surrounding the rectum; sometimes it develops in the course of acute and chronic inflammations and degenerations of the rectum; again it accompanies affections of the pelvis, or of the organs situated in the pelvis; at other times, like perityphlitis, it is one symptom of extensive metastatic inflammations. We also see periproctitis develop very often in patients who have consumption of the lungs and intestines. The cause of this complication is obscure, for the dependence of the inflammation of the connective tissue on a suppuration of caseously degenerated lymphatic glands has not been proved.

Acute periproctitis may end in resolution, but more frequently leads to abscesses which may subsequently perforate outwardly or into the intestine. Chronic periproctitis leads to decided thickening and induration of the inflamed connective tissue, but it also almost always ends in partial suppuration, and fistulous ulcers form and are difficult to heal.

At the commencement of acute periproctitis we find a hard, painful tumor in the perinæum, or in the vicinity of the coccyx. On introducing the finger into the rectum, we often recognize infiltration of the connective tissue by the feeling. The patient cannot sit up, and has the severest pain on defecation; if the inflammation terminates in suppuration, and the abscess perforates inwardly, the pain at stool increases, there is severe tenesmus, and, finally, purulent, stinking masses are evacuated per anum. This is the way that internal incomplete rectal fistulas are formed. If the abscess perforates externally, fluctuation occurs in the middle of the hard swelling in the perinæum, or near the coccyx, and, after the covering has been pierced, the above-described masses are evacuated. This process may cause an external incomplete rectal fistula. The symptoms of chronic periproctitis are usually obscure till the disease induces stricture of the rectum, and are hidden by the symptoms of disease of the mucous membrane, or other original disease. If abscesses form, there is severe pain along with the symptoms above described.

At first, we should attempt to bring the imflammation to resolution, particularly by the use of cold; later, we should apply cataplasms and fomentations, and open the abscess early to prevent perforation of the rectum or bladder.

## CHAPTER VII.

### HÆMORRHAGES AND VASCULAR DILATATIONS OF THE INTESTINE.

Etiology.—Hæmorrhages from the upper part of the intestinal canal occur from the same causes as hæmorrhages of the stomach. They most frequently result from excessive congestion of the portal circulation, such as occurs particularly in cirrhosis of the liver. In other cases, vessels of the intestinal mucous membrane are eroded by ulceration; such hæmorrhages occur during typhoid fever, dysentery, and in some few cases of consumption of the intestines. Lastly, there are hæmorrhages in the intestinal canal which must be referred to disease of the walls of the vessels, although the microscope shows no change of structure; among these are to be classed the intestinal hæmorrhages in yellow fever (?), scorbutus, etc.

Varicose dilatations of the hæmorrhoidal veins (blind piles, hæmorrhoids) and bleeding from the vessels of the rectum (bleeding piles) are among the most frequent of affections. It is not long since these were regarded as symptoms of a specific constitutional disease, hæmorrhoidal disease, and, according to the former opinion, they were the most favorable shape that the disease could assume; the case was far more serious if the disease were "misplaced," that is, affected the head, breast, or abdomen. This view has been generally given up, since it has been found how much the occurrence of venous dilatation and bleeding in the rectum is due to mechanical causes, and how little tenable is the idea of "misplaced hæmorrhoids." Nevertheless, the pathogeny and etiology are still somewhat obscure.

Obstruction of the circulation, which is the most frequent cause of congestion everywhere, must be regarded as the most common cause of hæmorrhoids, with which general name we shall designate the venous dilatations and hæmorrhages occurring in the rectum.

The escape of blood from the hæmorrhoidal veins may be caused:

1. By collections of fæces in the rectum, by tumors in the pelvis or the gravid uterus; and these are the most frequent causes of hæmorrhoids.

2. The escape of blood may be impeded by obstruction of the portal vein. Hence we shall mention hæmorrhoids as one of the most frequent symptoms due to the congestion in cirrhosis of the liver. Overfilling of the portal veins appears to have a similar effect, and perhaps this best explains the frequent occurrence of hæmorrhoids in drunkards. During digestion there is an increased flow of fluids from

the intestines into the intestinal veins; we know that the increased fulness of the portal vein, from this cause, obstructs the escape of blood from the splenic vein, and that consequently the spleen is enlarged every time that digestion goes on. But it readily follows that from excess in eating and drinking the fulness of the portal veins is increased, and is more permanent, and that consequently other veins which open into the portal veins may dilate, and from repeated excesses may remain dilated. This explanation is hypothetical, it is true, but it is not more so than other explanations that have been offered for the occurrence of hæmorrhoids from excess in eating and drinking.

3. The obstruction which impedes the escape of blood from the hæmorrhoidal plexus may lie beyond the liver, in the chest. Thus we often see hæmorrhoids develop in lung-diseases where the capillaries are compressed or atrophied; the patients considering them as the cause, not as the result, of their chest-disease. In the same way hæmorrhoids develop from heart-affections, along with other results of overfilling of the veins.

The above-mentioned obstructions to the circulation do not usually suffice to cause hæmorrhoids; their frequency is not proportionate to the amount of the obstruction; they are often absent when the escape of blood from the hæmorrhoidal plexus is greatly interfered with, while in other cases, where there is no perceptible obstruction except a temporary constipation, they become excessive. There is an analogous condition in the varicose veins of the legs of women during pregnancy; in some women the varicose veins appear during the first months, in others they do not occur even during the latter months, in spite of large amounts of liquor amnii, large children, or the most unfavorable position of the child. This goes to prove that the walls of the veins are less resistant in some persons than in others, and that this diminished tonicity of the walls of the vessels is most important for the occurrence of phlebectasias anywhere, and of hæmorrhoids in particular. This abnormal lack of resistance in the walls of the veins is in many cases congenital. The fact, that in certain families all the members for several generations suffer from hæmorrhoids, cannot be denied, and can only be explained by the supposition that a peculiar state of the vessels is hereditary. In other cases the want of resistance is undoubtedly acquired, and is probably induced by the disturbance of nutrition in the walls of the vessels from the chronic catarrh of the rectum. We have learned that dilatation of the veins is one of the anatomical appearances of chronic catarrh in all the mucous membranes, and hence can understand that the veins of the rectum, which from their position are peculiarly disposed to dilatation, should in a similar way become varicose from catarrh of the mucous membrane of the rectum. The variety of injuries that the rectum has to bear, the frequency of acute and chronic diseases of neighboring organs, in which the rectum is implicated, abundantly explain the frequency of chronic catarrh, and at the same time the frequency of relaxation of the hæmorrhoidal veins.

It is usually supposed that general plethora induces hæmorrhoids, and that bleeding piles are of critical significance in plethoric conditions. It cannot be denied that persons, who, particularly after they have attained their growth, consume more than is required for the support of the body, are often affected with hæmorrhoids; and also that gout, chronic catarrh, and other diseases, which are also frequent in such persons, are usually better after hæmorrhoidal hæmorrhage. Nevertheless, we should hesitate about referring either the hæmorrhoids or the other diseases, in these cases, to a general plethora, to an absolute increase of the contents of the vessels, as the permanent occurrence of such a state has not been fully proved; and there is good reason to suppose that the overfilling of the vessels leads to increased excretion till the disproportion has been removed. The changes that the blood undergoes from too great a supply of nourishment (abnormal concentration?) are not thoroughly understood, and hence the pathogeny of the diseases developing under such circumstances is quite obscure.

Hæmorrhoids are rarer in children than in adults; this is explained by the greater rarity of the above-mentioned obstructions to the circulation, and of chronic gastric catarrh, during childhood. On the other hand, it is evident how a sedentary life, the use of irritating food, the misuse of drastic purges, the frequent and clumsy use of enemata, may rank among the exciting causes of hæmorrhoids. If it be true, as is said, that piles are less frequent in women than in men, and in temperate climates than in hot ones, and that they are induced by excessive venery, we cannot so readily explain the fact by the causes above given.

Anatomical Appearances.—As the hæmorrhages from the upper part of the intestine are almost always capillary, their source can rarely be recognized on autopsy. Occasionally, after capillary hæmorrhages, a considerable extent of the mucous membrane is found suffused with blood, which is a sign that the hæmorrhage has taken place into the tissue of the membrane and not on its free surface. After hæmorrhages caused by ulcers in the intestine, coagula generally adhere to the ulcers which have bled, and the edges and base of the ulcer are suffused with blood. The blood is sometimes fluid, sometimes slightly coagulated, rarely red, but usually chocolate-brown, or transformed to an adhesive, black, tarry mass.

The varicosities of the rectum, which are termed blind piles, usually occur at the end of the rectum, above the sphincter and at the edge of the anus. The former are called *internal*, the latter *external* piles. At first the venous dilatation is diffuse and forms a thick blue net, afterward single varices appear, and not unfrequently the anus is surrounded by a wreath of the latter. At first the varices are small and have a broad base, they appear and disappear at intervals; later they may attain the size of a cherry or even become larger. As the internal varices are pressed through the anus, when the bowels are evacuated, and draw the mucous membrane after them, the latter often forms a pedicle for them and they remain outside of the anus; even then they sometimes appear tense, at others relaxed; but the sacs once formed never disappear. The appearance and structure of the hæmorrhoidal tumors change in the course of time. At first they are bluish and their walls are thin and delicate; if repeated chronic inflammations subsequently cause them to adhere to the mucous membrane, they lose their bluish look, and become hard and thick-walled. Not unfrequently neighboring varices coalesce, only rudiments of their partitions remain, and thus large, sinuous sacs are formed, into which several veins open. Occasionally a thrombus forms in the varices, filling them up and causing their obliteration and ulceration. Large varices which are extruded during defecation may inflame and even mortify from the pressure; in other cases there is inflammation and ulceration of the mucous membrane at the root of the hæmorrhoidal tumor and hæmorrhoidal ulcers result; in still other cases the inflammation attacks the surrounding connective tissue, and we have periproctitis, and, as a result of this, may have rectal fistula.

Bleeding piles result sometimes from the rupture of varices, but small hæmorrhages are mostly caused by overfilled capillaries.

According to *Virchow's* description, on anatomical examination of the rectal mucous membrane, we find it "relaxed, often in puffs and folds, slightly thickened, grayish-white; the submucous tissue is increased and relaxed; both are very vascular. It is usually covered with tough, whitish mucus, which chiefly consists of detached epithelium, with a mixture of mucus."

SYMPTOMS AND COURSE.—Hæmorrhages in the upper part of the intestine are, as above stated, symptoms of severe diseases, and must be described when speaking of these.

The description of hæmorrhoids given in the old text-books, and which still corresponds to the popular idea, distinguishes three groups of symptoms: 1. The local difficulties which are caused by the catarrh, the varices, and the hæmorrhages, "mucous or blind and bleeding piles;" 2. Periodical difficulties, both local and general, which precede the swelling of the varices and the hæmorrhage from the rectum, and are relieved by the latter, "molimina hæmorrhoidalia;" 3. Permanent difficulties, which indicate constitutional disease, or disease of some distant organ, but which are also relieved by the hæmorrhoidal bleeding, "misplaced hæmorrhoids," or, if hæmorrhages occur elsewhere, "vicarious hæmorrhoids."

We should strike the latter from the list of hæmorrhoids. If a venous abdominal plethora, dependent on cirrhosis of the liver, is improved by hæmorrhoidal bleeding, and the dyspepsia, flatulence, and hypochondria disappear for a time, this does not justify us in regarding these symptoms as signs of a hæmorrhoidal disease. We have just as little right to regard bronchial catarrh, or attacks of gout occurring in a plethoric person, as anomalous or misplaced hæmorrhoids, because these diseases remit after a hæmorrhoidal bleeding.

In regard to molimina hæmorrhoidalia, we must agree with *Virchow*, who regards it as a symptom of returning rectal catarrh. The patient has a feeling of burning and tension in the rectum, just as occurs in other mucous membranes in acute catarrh or relapsing chronic catarrh. There are also severe sacral and dorsal pains, which remind us of the headache in catarrh of the nose and frontal sinus. The general state of the patient is disturbed in the same way by catarrh of the rectum as it is by catarrh of other parts; the patients become relaxed, sluggish, and depressed. The inconveniences which the varices, swelled by the increased hyperæmia, cause, complete the picture of hæmorrhoidal hyperæmia. In many cases, at the height of the attack, there is a hæmorrhage, which has a favorable influence on the catarrh and the fulness of the varices, so that the patient feels relieved, or even free from all trouble. If, after a time, he be again affected with molimina, we cannot blame him for longing for the hæmorrhage that relieves him. If we can remove the catarrh and swelling of the varices in any other way, as by removing constipation, which has caused the increased congestion and hyperæmia of the rectum, the molimina disappear without a hæmorrhage.

The local difficulties that the hæmorrhoids excite vary with the number, size, and fulness of the varices. At first they are slight, the patients have the feeling of a foreign body in the anus, and pain only occurs when there is a hard stool. When the anus is surrounded by large varices, or when individual tumors have become very large, and are very tense, the patients have constant pain, cannot sit down, and even a soft passage gives them great suffering, which only disappears slowly, and which not unfrequently causes the patients foolishly to retain their passages. The pain becomes most severe when large varices are protruded through the anus, strangulated there, and become inflamed.

Hæmorrhoidal bleedings usually occur during defecation; if they are of capillary origin, only a small quantity of blood adheres to the fæces; if they come from ruptured varices, several ounces of blood are often lost. It is only in rare cases that there is sufficient hæmorrhage to cause danger.

The so-called mucous hæmorrhoids consist of the passage of the above-described catarrhal secretion; this is sometimes evacuated with the fæces, sometimes squeezed out of the rectum without any admixture of fæces. Frequently only the symptoms of mucous piles are present, and it is only subsequently that those of blind and bleeding piles occur.

From the usually prolonged action of the injurious influences causing them, it may be readily understood that the course of this disease is usually tedious. If the causes act only for a time, the hæmorrhoids may disappear forever, after lasting only a short time.

The variation of the symptoms of hæmorrhoids, after they have lasted a long time, has led to the most varied hypotheses. They have been compared to menstruation, and even the changes of the moon were claimed to have an influence on their course. The causes of the unpleasant feelings of the patient at one time, and his comfort at another, may often be discovered; the occurrence of constipation has obstructed the escape of blood from the rectum; or a debauch has caused an overfilling of the portal vein, and a consequent congestion of the hæmorrhoidal vessels; or they have been exposed to some other source of injury, which in them has not induced a nasal or bronchial catarrh, but has excited an increase of the rectal catarrh, because the rectum was the locus minoris resistentiæ. In other cases, the exciting causes cannot be discovered, but this also happens in the temporary exacerbations of other diseases, and so we are not justified in any adventurous hypotheses.

We hear a great deal about the dangerous effect of the arrest of habitual bleeding from hæmorrhoids. This belief is not altogether without grounds, but we should not consider the bleeding as Nature's attempt at a cure. The rectum is probably the part whose diseases have least effect on the organism, and patients in whom the rectum is the part soonest affected, when they are exposed to injurious influences, are better off than those whose stomach or bronchi are affected under similar circumstances. If they be affected with disease of one of the last-mentioned organs, when exposed to injury, we may deplore it, but if they have hæmorrhoids, they are just as correct in saying "all right" as one is who, having been exposed, begins to sneeze, and thereupon concludes he is only going to have a cold in the head, and not a worse disease. In cases where abdominal plethora, dependent on mechanical obstruction, is relieved by an occasional hæmorrhoidal bleeding, or where there is a remission of bronchial catarrh, or other disease, which usually exists in patients of forty years or upward, who lead a luxurious life, the cessation of the bleeding may prove serious. But, as in these cases, the relief depends solely on the loss of blood, and as this can be replaced by local blood-letting, the injury practically only occurs when the physician fails to see that the latter is indicated.

TREATMENT.—Where the repeated collection of hard fæces excites the catarrh and varices of the rectum, the causal indications require the regular evacuation of the bowels; but we should only use drastic purges, such as aloes and colocynth, when absolutely necessary, as we fear their irritant effect on the mucous membrane of the rectum, and should generally prefer flowers of sulphur or precipitated sulphur, which have long been used in the treatment of hæmorrhoids; sulphur is generally given in combination with tartrate of potash. One of the commonest prescriptions is ℞. sulphur. depur. ʒ ij, potass. bitart. ℥ ss, syrup. limonis, sacch. albi ana ℥ iij, M. ft. pulv. S. A teaspoonful two or three times daily.

If we do not succeed with this prescription, we may add some senna or rhubarb to it. Another popular way of prescribing sulphur is the pulvis glycyrrhiza compositus, of which a few teaspoonfuls may be taken during the day. Enemata are not generally advisable; for, even if carefully used, they are liable to irritate the rectum. Where cirrhosis of the liver, or diseases of the lungs or heart, cause the hæmorrhoids, we cannot usually fulfil the causal indications. In these cases, also, the administration of sulphur is advisable, so that a second evil may not be added to the first. As we have mentioned overfilling of the portal veins, from excess in eating and drinking, as among the causes of hæmorrhoids, so the casual indications require that such patients should not eat too frequently or too much. Finally, in those patients who, besides other troubles, have hæmorrhoids from excessive eating, we must lay down the most stringent rules. If there be a true plethora in such cases, it can only be explained on the supposition that, when the serum of the blood contains an increased amount of proteïn substances, particularly of albumen, it requires an abnormal fulness of the blood-vessels to cause the separation of the same amounts of fluid that are excreted with a normal fulness of the vessels. If the normal amount of albumen exists in the blood, we may regard it as proved that the amount of urine excreted diminishes with the increase of albumen in the serum of the blood. The above hypothesis also corresponds to the general belief of the laity and physicians: a man does not become full-blooded by eating or drinking *too much*, but by eating nourishing food and drinking spirituous liquors. Without entering into the question whether, in so-called plethora, there is actually an increase of the amount of blood, or only an increase of the blood-cells, or of the albumen in the blood (polycythæmia and hyperalbuminosis, *Vogel*), certain rules of life may be laid down for the affected persons which correspond to the physiological and practical view of the affection: 1. The use of proteïn substances must be limited; the patient should only eat a little meat or egg once a day, but should eat vegetables, fruit, rice, etc. 2. The consumption should be increased; the recommendation of long walks and energetic muscular exercise, and drinking plenty of water, which hasten the transformation of material, is just as rational as forbidding spirituous liquors, and tea and coffee, which seem to retard the transformation. 3. Such patients are very greatly benefited by saline purgatives, particularly by the moderate and continued use of glauber salts and chloride of sodium, as they occur in the waters of Marienbad, Kissengen, Homburg, Soden, etc. The use of the waters at Karlsbad requires great precautions, on account of the high temperature of the springs. If it be proved that, by this treatment, the blood grows richer in salts, and poorer in albumen (*C. Schmidt*, *Vogel*), there would be a rational explanation of its brilliant results in the treatment of plethora.

The *indications from the disease* do not present any further rules in cases where occasional moderate suffering is speedily relieved by the occurrence of spontaneous hæmorrhage; we content ourselves with fulfilling the causal indications as well as possible. But if the patients are tormented with severe molimina, which do not disappear after the removal of any existing constipation, we should apply from four to six leeches about the anus. After the leeches drop off, we should encourage the bleeding, by placing the patient on a night-stool, with a vase of warm water under it. The same proceeding is advisable when great fulness and excessive tension of the varices cause severe pain, or if hæmorrhoids are accompanied by painful tenesmus. We should let moderate bleeding continue, particularly when it promises relief from troublesome symptoms, and should only use cold or styptics when the loss of blood is considerable. Hæmorrhoids that have come down and been strangulated should be replaced by continued careful pressure with a bit of oiled linen, while the patient rests on his knees and elbows, with the body bent far forward. Inflamed hæmorrhoids should be covered with cold-water compresses, or bladders filled with cold water. We will not discuss the operative treatment.

With our view of hæmorrhoids, we cannot follow the ruling custom, and speak also of remedies for "bringing on suppressed piles." Luckily for the patients, the remedies recommended for this end, such as periodical abstraction of blood, warm sitz-baths, irritating suppositories, and the administration of so-called pellentia, rarely cause hæmorrhoids, while the periodical abstraction of blood attains the only object that it was sensible to aim at.

## CHAPTER VIII.

### NERVOUS AFFECTIONS OF THE INTESTINES—COLIC—ENTERALGIA.

AFFECTIONS of the sensory nerves of the mesenteric plexus—colic, in the strict sense of the word—are not by any means frequent. Analogy leads us to suspect their occasional occurrence from structural diseases of the ganglia and plexuses of the sympathetic nerve; but this has not been proved. The frequent occurrence of mesenteric neuralgia in hysterical females speaks for its reflex origin. Lastly, lead-colic is the most striking instance of a nervous affection caused by poisoning. In the latter case, however, there appears to be not a simple affection of the sensory nerves—a hyperæsthesia—but, at the same time, there seems to be a disturbance of the motor nerves—a hypercinesis—as the painful intestine is always contracted. The lead, whose absorption into the body causes lead-colic—one symptom of lead poisoning—is partly breathed in as fine powder, and partly absorbed from the intestinal and Schneiderian mucous membrane. Hence we find the disease among white-lead paint-makers, lead and silversmiths, painters, color-grinders, potters, type-founders, compositors, and others who work in an atmosphere loaded with particles of lead. The misuse of medicinal preparations of lead, the adulteration of wine and other liquors with sugar of lead, or by the accidental addition of lead to them, is at present a much rarer cause of lead-colic than those above mentioned. Still the celebrated colic of Devonshire, Poitou, and other epidemic and endemic colics, which very much resembled lead-colic, appeared due to poisoning from some drink containing lead, and not to poisoning by vegetable substances. In some rare but authentic cases, lead-colic has occurred from using snuff that had been packed in lead-foil. The predisposition to lead-colic is very varied, but among the predisposing causes we only know the great tendency to the disease left by a previous attack; all the other causes which are blamed, as increasing the predisposition to lead-colic, such as debauchery, drunkenness, etc., can hardly be denied, for they are found everywhere, when no other causes can be detected.

But by colic, in the wider sense, we understand, besides the nervous affections of the mesenteric plexus, all painful affections of the intestines which are not caused by inflammation or textural changes of the intestinal walls. So, among the symptoms of helminthiasis, we shall speak of colicky pains, just as we mentioned them when speaking of the premonitory symptoms of typhlitis stercoracea, and of contraction and obstruction of the intestines. But we have already distinguished the colicky pains which precede the inflammation from those which accompany and depend on it. The same cause which has to-day induced a colic, may to-morrow excite a colitis. We cannot always explain how these colics can induce increased excitability in the sensory nerves of the intestines; but we may suppose that the pains are always caused by irritation of the peripheral extremities of the intestinal nerves; so that this form of colic must be distinguished from the proper nervous affection of the intestine. The most frequent cause of colicky pain is, unmistakably, excessive distention of a portion of intestine, causing stretching of the walls of the intestine, and gases enclosed at some circumscribed part appear particularly to cause this distention. We may often clearly perceive that the gas is driven forward against the fæces, or some other obstruction, and, there arrived, it excites the most severe pains; and, in other cases, that the gases are driven by the contraction of the intestines, from one place to another, and, with their change of location, the position of the pain also changes. It is just as improbable that the pain in this *colica flatulenta* is caused by the irritation of the intestinal gases on the intestinal mucous membrane, as that it depends on the pressure from the contraction of the intestinal muscles on the nerves of the intestine. As the decomposition of the contents of the intestines is the most frequent cause of the collection of gas, it becomes evident that the diseases in which the contents undergo abnormal decomposition are often accompanied by the symptoms of wind-colic. This is particularly true of intestinal catarrh, which is excited by the passage of undigested food from the stomach into the intestine, or by the long retention of fæces. As in children, undigested and decomposing milk very frequently enters the intestines, *colica infantum* is an exceedingly frequent disease. If the decomposing substances be removed from the intestines before the intestinal mucous membrane is affected with catarrh, colic may be the sole symptom of the abnormal process. Just as colica flatulenta appears to be caused by a collection of gas in the intestine, *colica stercoracea* appears due to a distention of the intestines by fæces, and *colica verminosa* to a distention of the intestine by a coil of tape-worm, or a bundle of round worms. The abdominal pains following the employment of drastics or injurious ingesta are also usually described as colic, but the changes in the secretions of the intestine after the use of these medicines, or after eating unripe fruit, and many other substances, tend to show that there is slight inflammation, which is of short duration, and disappears with the removal of the injurious substances. We may well compare these pains to those that result when a sinapism is applied to the skin, and which disappear as soon as the sinapism is removed. Perhaps some cases of colica verminosa also belong here, particularly of those where the attacks of pain are followed by the discharge of large mucous masses—so-called worm-nests. In the painful and long-continued attacks of colic occurring after exposure of the skin, particularly that of the feet and abdomen, to cold, the muscular coat of the intestines appears to suffer in the same way as muscles elsewhere do in rheumatic affections; hence the affection is well-named *colica rheumatica*.

SYMPTOMS AND COURSE.—*Romberg* describes *neuralgia mesenterica* as follows: "There are attacks of pain spreading from the navel over the abdomen, alternating with intervals of ease. The pain is tearing, cutting, pressing, most frequently twisting, pinching, introduced and accompanied by peculiar bearing-down pains. The patient is restless, and seeks relief in changing his position, and in compressing the abdomen; his hands, feet, and cheeks are cold; his features are pinched; the wrinkled brows and contracted lips betray his agony. The pulse is small and hard. The skin of the abdomen is tense, whether puffed up or drawn inward. There are often nausea, vomiting, and desire for stool; sometimes there is also tenesmus. There is usually constipation, but sometimes the bowels are regular, or even too loose. Such an attack may last from a few minutes to several hours, relaxing at intervals. It ceases suddenly, as if cut off short, and there is a feeling of the greatest relief. The course is periodical, but less regularly so than in other neuralgias."

Lead-colic is almost always preceded by the symptoms of lead-poisoning. The patients are thin and badly nourished, their skin looks dirty and earthy, the gums are dark, almost slate-gray, the teeth themselves discolored, and the breath bad; the patients have a sweetish, metallic taste in the mouth. Then there are periodical pains, which are at first dull, and extend from the epigastrium toward the back and extremities. The pain soon becomes more severe, so that, during the attack, the patients moan and groan, toss themselves about on the bed, or else leave the bed in despair, and do the most foolish things. At the same time, the pulse becomes much slower, the voice is lost, and strangury, nausea and vomiting often occur, showing that the abnormal excitement of the intestinal nerves has spread to other nerves. There is almost always obstinate constipation, and, in spite of the most powerful drastics, eight to fourteen days may pass before the evacuation of a small amount of dry, hard, spherical fæces. The abdominal walls are in a very peculiar state; they are strongly contracted, and the belly seems as hard as a board, and is drawn in. With few exceptions, the course of the disease shows a distinctly remitting type, so that paroxysms of the severest torture are varied with intervals of comparative ease. The duration of the disease varies: under suitable treatment, the first attack of lead-colic usually subsides in a few days or weeks; after repeated returns, the attacks may last for months. When the disease terminates in recovery, this may occur suddenly or gradually; the pains subside, there are free evacuations, and strength soon returns. The cure is often incomplete, and, after the lead-colic has been removed, symptoms of chronic lead-poisoning may remain. The disease rarely causes death, and, even in such cases, the patients do not die of the lead-colic, but of some of its complications.

The above description of a mesenteric neuralgia will also answer for that of colic in the wider sense. *Henoch* is correct in saying that the quality of a pain is the same, whether caused by the irritation of a nerve at its peripheral expansion, at its origin, or during its course. In colica flatulenta, and other colics of this class, the pain may become very severe, and then its intensity is depicted in the changed appearance of the patient; he is near fainting, the body is covered with cold sweat, the visage is pale and distorted, the pulse small; occasionally there are also nausea, vomiting, strangury, and other similar circumstances. We should know these symptoms, so as not to be deceived and unnecessarily worried. We can often hear and distinctly feel, on the abdomen of the patient, that the gas is freed from its imprisonment, and passes into other parts of the intestine. This is an important event; with its occurrence the pain often disappears instantly. In other cases, there is no improvement till the patient has a passage, and the fæces, which distended the intestine, or behind which the gases were collected, are evacuated.

TREATMENT.—In the neuralgic form, the *causal indications* may require treatment of the uterine disease that has induced the neuralgia. In lead-colic, the attempt has been made to fulfil the causal indications by chemically precipitating the lead that has been taken into the body. With this view, sulphuric acid and sulphates, particularly alum and glauber salts, have been prescribed. Although we can do little by this, or any other treatment, to remove lead-poisoning, we may do much to prevent its occurrence. With this view, we should avoid the use of lead in making pipes and vessels for conducting or holding water and other fluids for drinking. The workers in founderies, and other places where particles of lead fill the air, should bathe and wash carefully, and change their linen frequently; they should not eat in their workshop, and the latter should be very airy, and well ventilated. Zinc-paint should be used, instead of lead-paint, for painting doors and windows, and it should be a penal offence to pack snuff in sheet-lead.

In colic caused by something abnormal in the intestines, evacuants are indicated, particularly those which do not, like the drastics themselves, cause colicky pains. The internal administration of castor-oil, and the use of enemata, are most advantageous. If exposure of the feet or abdomen were the cause of the colic, the causal indications require a diaphoretic treatment; and the popular aromatic teas, the bottles of warm water and warm stones applied to the abdomen, are very suitable in these cases.

In all forms of the affection, the indications from the disease require the administration of narcotics, particularly of opium. In the neuralgic form, the action of opium is explained by its anæsthetic effect. In colica stercoracea, flatulenta, etc., there seems to be also a second action. The contractions of the intestinal muscles, which drive the gases and fæces toward certain parts of the intestines, or confine them there, are removed by the use of opium, and thus the contents are enabled to spread out over large portions of the intestines. In colica flatulenta and stercoracea, warm teas of camomile, peppermint, and valerian, drunk by the cupful, or used as enemata, have great reputation, as have also some other carminatives, and long and continued friction of the abdomen with warm oil. Opium is the most effectual remedy against lead-colic, and is used, even by the homœopaths, in full doses, in this disease. We should not be afraid of using it, under the impression that it will increase the constipation already existing. There is no remedy more successful than opium in relieving the constipation in lead-colic. This seems to favor the idea that, in that disease, besides the hyperæsthesia, there is a spasmodic contraction of the intestine, and that this causes the constipation. (*Romberg* considers this hypothesis improbable, as we cannot believe in a spasm lasting a week, and he considers the immobility of the bowels to be due to the pain, just as in sciatica the movements of the leg affected are restricted.) But, although opium is the most important remedy for the constipation, and is of more benefit than laxatives, when it is given alone, still we should not neglect to use them *with* opium. Recently, croton-oil is the cathartic most frequently given in lead-colic. In most cases, we shall have a good result from the administration of ½—1 gr. opium, three times daily, and every two hours one tablespoonful of a mixture of croton-oil (gtt. iij) and castor-oil ( ℥ ij). Warm baths, narcotic cataplasms, and the alternate use of laxative and narcotic enemata, aid this treatment. Besides this simple treatment, and slight modifications of it, consisting in the use of other laxatives, such as epsom salts, senna, calomel, and a bolder or more careful use of opium, there is a series of complicated methods, among which the treatment at La Charité is particularly celebrated. In all these methods of treatment, among the numerous remedies administered, there are always laxatives and opium, and their effect seems to depend on these remedies.

## CHAPTER IX.

### WORMS IN THE INTESTINAL CANAL—HELMINTHIASIS.

The worms most frequently found in the human intestines are the tænia solium, the tænia mediocanellata, the bothriocephalus latus, the ascaris lumbricoides, the oxyuris vermicularis, and the trichocephalus dispar.

The *tænia solium*, the long tape-worm or chain-worm, is from ten to twenty feet long, yellowish-white, thin and round at its anterior end, and broader and flatter posteriorly. It is divided into head, neck, and body, the latter consisting of many hundreds of links. The head forms a blunt, square, bulbous enlargement; it consists of a slightly-prominent conical snout, which is surrounded by a double row of hooks, and farther back by four round suckers symmetrically placed. The neck is very slender and about half an inch long. Then come the youngest links, which are scarcely quarter of a line broad, while the terminal and at the same time the oldest links may be half an inch wide or even wider. The individual links, which in shape remind us of a pumpkin-seed with the ends cut off, have different structure according to their age. The younger ones have a simple, slightly brownish-yellow median canal, with short lateral off-shoots, the first indications of sexual organs. The older links have a small prominence at the edge, sometimes on one side, sometimes on the other, but not regularly alternating, from which the sickle-shaped penis projects, and into which the tortuous seminiferous tubes and the oviducts empty. The interior of the older links is almost wholly occupied by a uterus branching out to both sides or by the ovary. In the oldest links, the latter is filled with eggs, and the small embryos with their six small hooks may often be distinctly recognized. Vessels start from a vascular ring within the head, and run down the side of the links; according to some observers, these communicate by transverse canals. Thus far no other organs have been observed in the tænia.

The tænia solium inhabits the small intestine, but may enter the large. There is usually only one, but there may be two or more, in one person. Tænia solium occurs in Europe, America, Asia, and Africa; and, very curiously, it is not found, except in Switzerland, in countries where the bothriocephalus latus occurs.

Until within a few years, the *tænia mediocanellata* has been confounded with the tænia solium, and in fact its individual links are very similar to those of the tænia solium. They have the lateral sexual openings, but the links are broader and thicker; and the sexual organs are more fully developed, and more branched than in the links of the tænia solium. The difference in the head is more decided. The head of the mediocanellata has neither snout nor the row of hooks, but is flat, with four large suckers. This species has remained unknown so long, because we rarely succeed in expelling the head, and it has been customary when we found links where the sexual openings were on the side, to consider them as cases of tænia solium, and when the links had the openings in the middle, to regard them as bothriocephalus latus.

The *bothriocephalus latus*, the broad tape-worm, resembles tænia, but may readily be distinguished from it. In its head, instead of the snout, the hooks, and suckers, we find only two lateral slit-shaped fossæ. The neck is scarcely to be seen. The breadth of the links is much greater than the length; they are about in the proportion of three to one. The most important distinguishing mark between single links of the two varieties is the position of the sexual openings. In the bothriocephalus these are not to the side but in the middle of the links, so that in them we may speak of a belly and back. The bothriocephalus also inhabits the small intestine. It occurs in eastern Europe as far as the Vistula, and in Switzerland it is found with the tænia solium.

The *ascaris lumbricoides*, the round worm, is cylindrical, pointed at both ends, from six to twelve inches long, and from two to three lines thick. The body is so transparent that we may see the intestinal canal, extending from one end to the other, and the sexual organs. There is a circular depression behind the head; the latter has three small elevations, between which lies the mouth. The sexes are in different individuals. In the female there are large ovaries and oviducts; the male is smaller and somewhat crooked at its tail-end, which contains the long, tortuous, seminiferous tubes and the testicles. At the tail-end of the male we may see the hair-like, sometimes doubled, penis. In the upper third of the female is a fissure six to eight lines long, the opening of the sexual organs. The round worm inhabits the small and large intestine, but makes excursions in various directions and may enter the stomach, œsophagus, or even the larynx. From the duodenum it occasionally makes it way into the ductus choledochus. It does not seem probable that it can perforate the intestine; but if the intestine be perforated by any disease, we not unfrequently find round worms in the cavity of the abdomen. They often occur in incredible numbers.

The *oxyuris vermicularis*, thread or maw-worm, is a small worm about as thick as twine. The males are rare, about a line to a line and a half long, and rolled up at the tail-end. The females are larger and straight, or only slightly bent. The head of the thread-worm is enlarged by wing-like attachments. The penis is at the tail-end of the male. The sexual opening of the female is near the head. The usual seat of this worm is the lower part of the intestine, particularly the rectum, but they may even enter the lower part of the small intestine. They often crawl out of the anus and enter the vagina, etc.

The *trichocephalus dispar*, the hair-headed or whip-worm, is about an inch and a half or two inches long; the posterior part is quite thick, the anterior hair-like. In the male, which is the smaller, the posterior part is wound into a spiral, and has at its end the hook-shaped penis surrounded by a bell. The female is thicker and straight, and its posterior end is full of eggs. The trichocephalus inhabits the large intestine, particularly the cœcum.

*Development of Intestinal Worms, and Ætiology of Helminthiasis.*—The time for believing in spontaneous generation, and in the formation of intestinal worms by a collection and alteration of intestinal mucus, has passed. The parasites living in the intestinal canal originate from eggs, and have reached the intestines in that state, or one further advanced. Of the tænia solium and tænia mediocanellata alone, we more accurately know the mode of development. The last links (proglottides) of the tape-worm, which contain the ripe eggs, occasionally drop off, and are evacuated. In order to develop further, the embryos from the eggs must enter some other animal. If they are swallowed by some animal, they pass from the intestines into the tissues of the body, till they find a suitable place; then they throw off the little hooks, and a neck and head (scolex), resembling those of the tape-worm, grow from their wall. At first the scolex is enclosed within the embryo; it subsequently becomes free, and the swollen body of the embryo hangs to it like a bladder. At this stage of development the scolices constitute the parasites known as cysticerci, or bladder-worms; the most common variety of them, those found in swine, called cysticercus cellulosus, is the scolex of tænia solium. If this cysticercus enters the intestines of a human being, it becomes attached to the wall, drops the bladder-like tail, forms links, and becomes a tape-worm. Tænia mediocanellata develops in the same way when a cysticercus living in *beef* enters the intestines of man. The scolices of bothriocephalus latus are unknown, as are also the first stages of ascaris lumbricoides, oxyuris vermicularis, and of trichocephalus dispar; but it is certain that the young worms are not directly developed from the eggs of worms existing in the intestines. Hence we must suspect that the young of these worms also are taken into the body with the food.

Our ideas of the etiology of helminthiasis have been totally changed by the discoveries of recent times; most causes to which it was formerly attributed are now recognized to be without effect. It even appears improbable that any decided change of the intestinal mucous membrane, or any peculiarity of the contents of the intestines, is necessary for the development and future life of the worms. The etiology of tænia solium is frequently discoverable. *Küchenmeister* found young tænia in the intestines of a decapitated criminal, to whom he had administered cysticerci a few days before death. Of the animals whose flesh we eat, swine are the most apt to have the scolex of tænia solium. It also occurs in the meat of the goat, and, although more rarely, in beef. In Jews and Mohammedans, who eat no pork, tænia are very rarely found; and, while in Abyssinia almost every one has tape-worm, the Carthusian fathers, who eat only fish, remain exempt from them. Tænia are much more frequent in regions where pork-raising flourishes, while they are rare where swine are scarce. Cysticerci cannot withstand boiling, roasting, or smoking, and tape-worm never results from the use of measly meat prepared in these modes. On the other hand, they are most likely to occur in persons who eat or chew raw flesh, or put knives, soiled with cysticerci, in their mouths, as is not unfrequently done by waiters, cooks, and butchers. The latter may greatly aid the spread of tape-worm by cutting the sausage or ham that they sell with a dirty knife, as these articles are often eaten without further cooking. The practice of giving badly-nourished children raw shaved meat to eat is not devoid of danger, for cases have occurred where children have undoubtedly acquired tape-worm (tænia mediocanellata) in this way.

The supposition, that the use of meat, containing trichina spiralis, led to the development of trichocephalus dispar, has been disproved. If, as has been supposed, ascaris and oxyuris are most frequently found in persons who subsisted chiefly on amylaceous food, this might be explained by the observations of *Stein*, who found entozoæ in weevils. It is possible that, by using bad flour, eggs or larvæ of ascaris or oxyuris may reach the intestines.

SYMPTOMATOLOGY.—The symptoms excited by intestinal worms vary greatly with the peculiarities of the person affected. Frequently there are no signs till worms, or fragments of worms, are passed at stool. This is chiefly true of tape-worms. Many patients with tænia, or bothriocephalus, enjoy the best health, have neither stomach-ache nor any reflex symptoms, and the links that pass away, from time to time, alone call attention to their disease. Frequently it is difficult for the physician to recognize the dried proglottides that are brought to him wrapped in paper. In other cases, the patients complain, from time to time, of severe pain in the abdomen, which they describe as twisting and turning (but, perhaps, they may not do so until they know they have a tape-worm); they double themselves up, or press the abdomen against some hard substance, have nausea, and, occasionally, vomiting. The secretion of saliva is almost always increased, so that the saliva flows out of the mouth. The attacks above described, particularly when they occur after the use of herring, onions, horse-radish, or large-grained fruits, are by the laity considered as a certain sign of worms, and should excite in the physician also a suspicion of tape-worm; but there is no certainty about it till links of the tape-worm have been passed, either spontaneously or after the use of laxative or anthelmintic remedies. In other cases, the presence of the parasite is not so well borne; occasionally, particularly after eating salty or spiced food, there is diarrhœa; the patient becomes weak, pale, and thin. This occurs particularly in persons previously debilitated, especially in children and young girls. Lastly, the irritation of the intestines, caused by tape-worm, may be reflected to other nerves; but the frequency of nervous affections from this cause has been much exaggerated, and this exaggeration has led to numerous errors. The sensation of tickling in the nose, which induces the patient to rub and pick that organ, the dilatation of the pupil, squinting, grating the teeth, and other unimportant and more isolated disturbances of innervation, are rather attributed to the round worm, while the tape-worm is blamed with severer and more extensive nervous affections, particularly epilepsy and St. Vitus's dance. We may entertain a faint hope that epilepsy, occurring without any perceptible cause, depends on irritation from worms, but we should avoid the belief that the passage of links of tape-worm from an epileptic patient proves that the disease depends on the presence of the worm, and will disappear with its removal. Cases where this occurs are very rare, compared to those where the epilepsy remains the same after the removal of the tape-worm as it was before.

As a rule, the presence of round worms in the intestines excites no symptoms. They are so very common that, if the intestinal canal were much irritated, and nutrition greatly impaired by their presence, there would not be so many healthy and blooming children. If there be a large number of them in the bowels, they may curl up together, and form an obstruction, as hard fæces sometimes do, or cause colicky pains; if they do not again uncurl, or cannot be removed by cathartics, they may induce the symptoms of ileus. In other cases, active movements of the round worms seem to excite abdominal pains, similar to those caused by tape-worms. The causes of this disquiet of the worms is unknown, and *Küchenmeister's* suggestion, that perhaps the round worms become more lively during coitus, appears to be meant as a joke. To decide from a pale, cachectic appearance, and from the nervous symptoms above mentioned, that children are suffering from round worms, is an error, even more common than it is to diagnosticate a tape-worm in an adult who has the same symptoms. If such children pass round worms from the bowels, or vomit them, it is too common to consider the diagnosis as verified, and to neglect a more careful examination, and when, too late, the discovery is made that the symptoms do not depend on the worms, but on hydrocephalus, or some other dangerous disease. The same is true of worm-fever. Occasionally round worms may induce intestinal catarrh, and slight febrile symptoms, but, in most cases, the worms passed have nothing to do with the fever ascribed to them. If the round worms enter the stomach, they occasionally cause great discomfort, anxiety, and nausea. The patients cannot describe how sick at the stomach they are, and the physician may be perplexed till the worm is vomited, and the riddle thus solved. In other cases, the presence of the worm in the stomach and œsophagus causes so little difficulty that it may crawl out of the mouth during sleep, without awaking the patient. If, in its wanderings, it enters the larynx, there will be spasmodic contraction of the glottis; children have even suffocated as a result of this accident. If the round worm finds its way into the ductus choledochus, there may be biliary obstruction, and, if it pass on still farther into the gall-ducts of the liver, there may be partial hepatitis; but we can rarely interpret the symptoms correctly, and refer them to a stray worm.

When the *oxyuris* approaches the anus, or crawls out of it, its incessant motions cause a very troublesome itching. This usually increases late in the evening and during the night, and interferes with sleeping. Besides the itching, there is usually an incessant desire to go to stool. In the evacuations, which are often mixed with mucus, the worms continue their snaky movements. If they crawl over the perinæum to the vulva and vagina, they cause annoying prickling and itching there also. The irritation from the worms, and from the rubbing that they induce, may cause catarrh of those parts, and the frightened mother brings the child to the doctor, saying "it has the whites already." In such cases a careful examination quiets all fears. The *trichocephalus dispar* causes no symptoms.

TREATMENT.—The prophylactic treatment for tænia solium follows, as a matter of course, from what has been said. No pork should be eaten that has not previously been subjected to the procedures by which any cysticerci in it would be killed. Cooks should be forbidden to taste raw sausage, or to hold the kitchen-knife in the mouth. Butchers should be instructed not to cut sausage or ham with the knife they use for raw meat. We can offer no prophylactic treatment for the other varieties of worms, as we do not know their mode of origin.

Of the numerous remedies which were formerly used for removing tape-worm, we now only employ male fern, pomegranate-rind, kooso, and oil of turpentine [pumpkin-seeds].

Male fern—*Radix filicis maris*—appears to be chiefly efficacious against bothriocephalus, and often fails when given for tænia solium. Half a drachm or a drachm of the powdered root is given at a dose, and two or three such doses are taken in the morning, fasting, or at bed-time. A few hours later, or, if the powder be taken at bed-time, the next morning, we give a sharp laxative of gamboge, scammony, or calomel, or an ounce or more of castor-oil. The ethereal extract of male fern, which is usually made into pills, with equal parts of the powdered root, and given in doses of a scruple or half drachm, divided into two portions, is more certain and more easily taken. Male fern enters into most of the numerous and complicated worm-medicines, which are of late more and more neglected.

Pomegranate-rind—*Cortex radicis punicæ granati*—when fresh, appears to be one of the most certain remedies against tænia solium. We pour a pint or two of water over two or four ounces of it, and, after macerating for twenty-four hours, boil it down to one-half. This decoction is generally divided into three doses, and used in the morning, fasting, and, although very usually efficacious, it is occasionally vomited by the patient, and always causes excessive pain in the abdomen for hours. I can urgently recommend that, before using the decoction, the simple maceration should be tried; this is also made from two or four ounces of the rind. This maceration acts much more mildly; the patients suffer scarcely any, and after its use I have frequently seen one tape-worm passed, and, in one case three, with their heads, were passed. If the maceration fails, the decoction may be tried in a few days. After the exhibition of pomegranate-rind, the worm usually passes, unbroken, and is often rolled into a ball. If it be not passed in from one to three hours after the last dose, we may give one or two ounces of castor-oil. *Küchenmeister* recommends making an extract from four or six ounces of pomegranate-rind, and adding this, with from four to six ounces of hot water, to a scruple or half a drachm of ethereal extract of male fern, and four to six grains of gamboge. Two cups of this mixture taken, with an interval of three-quarters of an hour, are said to expel the worm. If this do not result in an hour and a half, the third should be administered.

Koosso—the dried and powdered flowers of *Brayera anthelmintica*—a remedy recently introduced from Abyssinia, has not fulfilled the expectations entertained of it; at least, the brilliant results attested by some observers have not been attained by others. From two drachms to half an ounce may be macerated in water, or made into an electuary with honey, and given in two doses, with an interval of half an hour, in the morning, after a cup of coffee has been taken. If nausea occur, we may give some lemon-juice. If the patient do not have a passage in three hours, we may give a dose of castor-oil or senna.

Although oil of turpentine is among the most certain remedies for tape-worm, it should only be used in case of necessity, not only on account of its disagreeable taste, but because in the requisite doses it is apt to irritate the urinary organs. One or two ounces of oil of turpentine alone, or mixed with honey or castor-oil, or in emulsion, are to be given in one dose, at bed-time.

It is best to use any of these remedies at times when some of the links of the tape-worm have been passed spontaneously; but it is quite unnecessary to delay treatment till certain phases of the moon, when, according to popular belief, the worms may be more readily dislodged. We should employ some preparatory treatment: let the patient live moderately, keep his bowels open with castor-oil, and let him live for a few days almost exclusively on herring, ham, onions, and other salty and spicy food. Instead of the above, the patient may eat freely of wild strawberries, huckleberries, etc., as the numerous seeds of these fruits appear to sicken the worm (*Küchenmeister*). The cure cannot be regarded as perfect till we find the head of the animal; nor must we forget that there may be more than one tape-worm in the intestines. *Kameela*, a powder obtained from the capsules of *Rottera tinctoria* (ʒ ij—iij, rubbed up with water), *cortex musenæ* (℥ i—ij, with honey), *radix ponnæ* (ʒ j—ij), and a few other medicines, have been more or less lauded as remedies for tape-worm, but after repeated trials no one of them has proved peculiarly efficacious.

For *ascaris lumbricoides*, *semina cynæ vel santonici*, the buds of *Artemisia contra*, justly enjoy the best reputation. The practice of giving an electuary, made of the powdered seeds of worm-seed, jalap, valerian, honey, and other substances, by which children were formerly tortured several times a year, as well as its exhibition in the shape of worm-chocolate or cakes, is now almost displaced by the more certain and agreeable preparations, such as the ethereal extract, and particularly santonin. Of the former we may give a child gr. v—x during the day, of the latter, gr. iij—jv. Apothecaries often keep troches of santonin, containing gr. ss—j each, which taste pleasantly. *Küchenmeister* advises dissolving santonin, gr. ij—iv, in castor-oil ℥ i, and giving a teaspoonful of this solution every hour till it acts; he had still better results from the santonate of soda, in doses of gr. ij—iv, given morning and evening for several days. A laxative should always be given after the use of worm-seed, or its preparations. Other anthelmintics for expelling round worms may be dispensed with.

Enemata suffice to drive the *oxyuris* from the rectum. Even injections of cold water, with a little vinegar, are very efficacious; but they should be very large, so as to reach any of the worms that may be up in the sigmoid flexure, and they should be used for a long time. In obstinate cases we may add a weak solution of corrosive sublimate (gr. ¼ to ℥ ij) to the enema.

(In my last edition, trichiniasis was considered at this place, but in this one I shall speak of it among the infectious diseases; the reasons for this will be stated when speaking of its etiology.)

## CHAPTER X.

### GASTRIC FEVER, CATARRHAL AND BILE-FEVER.

MANY physicians, particularly among the Germans, describe as *gastric fever* a disease running an acute course, in which high fever is only accompanied by dyspeptic symptoms, and generally by diarrhœa, while there are usually no symptoms that would indicate severe disease of any important organ. Celebrated authorities, particularly those clinical observers who have developed in hospital, and have had only hospital practice, consider all cases of so-called gastric fever as mild cases of typhus. I cannot at all agree with this view. Every physician in private practice often has the opportunity of seeing, after errors of diet, without any suspicion of infection, symptoms of variable duration, which exactly answer to those of gastric fever. If this be so, even where we can find no error of diet, we must be careful about inferring that there is an infection, and must acknowledge the possibility that catching cold, atmospheric and telluric influences, and other sources of injury, may excite a similar set of symptoms. But I will not attempt to deny that numerous slight cases of typhus are diagnosticated as gastric fever.

As a rule, gastric fever begins with several slight chills, rarely with one severe one. The pulse quickly rises to 100 or more. According to the few observations that have been made, the temperature is sometimes normal, in other cases it is decidedly increased; it may reach from 102° to 105°. The constitutional disturbance is very marked. The faintness is so great that the patient remains in bed; the limbs, particularly at the joints, pain "as if they would burst." The insupportable headache is usually increased by laying the head on a feather pillow, while it is occasionally relieved by binding a towel firmly around the head. The patient does not sleep at all, or is disturbed by dreams. The symptoms of disease of the stomach or intestines vary. Usually the appetite is lost, the tongue coated, the taste slimy or bitter, the breath is bad, the patients complain of a feeling of pressure and fulness in the epigastrium, and are sensitive to pressure there. There is also eructation of gases and fluids, usually acid products of abnormal gastric digestion. Occasionally there is repeated vomiting. At first there is usually constipation; but later, particularly when the disease is protracted, there is diarrhœa, preceded by more or less colicky pain; the stools are fluid, and colored green by bile, and are sometimes mucous.

Occasionally these symptoms pass off quickly, and the patient, who is one day in a sad plight, feels quite well the next (ephemera). At the same time herpetic vesicles not unfrequently come on the lips. We should not consider this a distinct disease, a febris herpetica. Herpes labialis accompanies gastric fever as often or perhaps oftener than it does pneumonic or intermittent fever, and has the same significance in the former disease as in the latter. But the disease does not by any means always terminate in one day; it often continues several days, but rarely longer than a week. In persons who do not bear well the feverish increase of temperature, or the consumption caused by the increased development of heat (we have frequently said that individual peculiarities vary greatly in regard to this), there is great depression, the mind is affected; instead of dreams, the patient has delirium; and, if at the same time the tongue become dry, the similarity with typhus is very great. It often happens that the true nature of the case is only explained at the sixth or eighth day, by the sudden improvement and the rapid convalescence.

In consideration of the difficulty of diagnosticating gastric fever from a commencing typhus, it is advisable to be very guarded in diagnosis and prognosis during the first week. It would be very dangerous for the reputation of the physician, if, after he has pronounced the disease to be gastric fever, and promised improvement from day to day, it should develop with all its terrors in the second or third week. But it will compromise the doctor just as much if the supposed nervous fever terminate in cure at the end of the first week, and the patient be able to walk out a few days later. Even the laity no longer believe that under certain circumstances "gastric fever" may become "gastric nervous," and this again develop into "nervous fever." They know that these two diseases are of different nature from the first. In making a differential diagnosis during the first week, great attention should be given to the etiology. If there have been injurious influences that could cause gastric and intestinal catarrh, in doubtful cases the presumption will be in favor of gastric fever. If, on the other hand, there have been numerous cases of typhus in the city or vicinity, and no errors of diet can be discovered as causes of the disease, we should suspect typhus. Secondly, the increase of the bodily temperature is not so regular in gastric fever as in typhus. Thirdly, catarrh of the finer bronchial tubes, with cough and sibilant rhonchi, indicates typhus rather than gastric fever, although bronchial catarrh may occur in the latter also; such cases are usually termed gastro-catarrhal. Fourthly, an eruption of herpetic vesicles about the mouth almost certainly excludes typhus. Fifthly, and lastly, a perceptible enlargement of the spleen, and the appearance of roseola spots on the upper part of the abdomen at the end of the first week, speak against gastric and in favor of typhus [typhoid] fever.

Cases occur where from the great general disturbance the gastric symptoms are thrown so much in the background, that we may doubt whether the disease is venting itself in the intestinal canal, and whether the fever and the symptoms caused by it can really be regarded as symptoms of gastric and intestinal catarrh. It is such cases that have led to the formation of the class called simple (essential) fever, *fièvre simple continue*, or synocha. I doubt the propriety of believing that fever can occur as the sole effect of the action of any injurious influence on the body. It seems much more probable that even in such cases there is structural change, which we cannot at present discover, in some organ or other. I consider this hypothesis justifiable from the well-known fact that, in numerous cases of pneumonia, erysipelas, and severe nasal and bronchial catarrhs, the fever and great general disturbance appear before the local symptoms. It is very difficult for me to believe that here also there is at first an essential fever, to which a local affection is subsequently added; and the more so, as, after this occurs, the fever and local disease keep step, and the former disappears when the latter has run its course. Now, if the delicate organic disease do not reach so high a grade as to cause evident functional disturbances, according to my hypothesis we have the state usually called essential fever. In any fever there is slight dyspepsia; and simple want of appetite, slightly-coated tongue, etc., do not justify us in designating a febrile affection as gastric fever.

Even more decidedly than in the case of gastric fevers does *Griesinger* say that the rare but very regular and characteristic disease called catarrhal fever, febris pituitosa, is also a typhus disease, which has, it is true, a peculiar and unusual course. I do not know whether *Griesinger* himself has made a large number of autopsies in cases of this disease, or whence he derives his authority for saying "that we find open or even cicatrizing intestinal ulcers in these cases." The long duration of the disease, the slightness of the fever, the great extent of the catarrh, the excessive production of mucus, and other points, decide me to doubt the correctness of *Griesinger's* belief till I have learned the facts on which he bases it. The description which I shall now give of the symptoms and course of catarrhal fever, I take partly from my own observation, partly from the excellent description of this disease, given in *Schönlein's* lectures, and which exactly corresponds with my own observation.

This disease does not begin with frequent pulse, pain in the limbs, severe headache, and restlessness, as gastric fever does. The pulse is usually moderately increased, the temperature slightly elevated, but the patients feel very dull and heavy, are apathetic, constantly sleepy, and disgusted at all food. If the patient be compelled to eat something, he soon has a distressing feeling of fulness; then vomiting occurs, and the food is thrown up, enveloped in large quantities of tough mucus. The accompanying oral and pharyngeal catarrh is also peculiar: the coating of the tongue is, at first, thick and yellowish; teeth and gums, palate and pharynx are covered with tough mucus; later the whole epithelial covering of the tongue is often thrown off, and it then looks red, like a piece of raw meat, or as if coated with varnish. In the morning, especially, the patients raise so much mucus, by spitting, hawking, vomiting, and coughing, that a spittoon will hardly contain it all; quantities of mucus are mixed with the undigested food, in the passages from the bowels, while the urine contains a mucous deposit. Even in the subsequent course, the fever remains moderate, and has sometimes a remittent, sometimes a continued type. The patients become very feeble; their apathy increases so, that while they do not sleep, they lie without any interest in their own state, or in things about them. If the disease begins to mend, which frequently does not occur till the third or fourth week, the production of mucus gradually ceases, the appetite slowly returns, the pulse becomes very sluggish, and the exhausted patients do not recover strength for a long time. The slightest cause induces a relapse; then the process begins anew, and months may pass before a perfect cure, or, in weak, decrepit persons, death may result.

It is difficult to determine what disease the older physicians meant by bilious, or gall-fever. I hope, however, by my observations during the last few years, to have arrived at a better understanding of those fevers accompanied by icteric symptoms. I no longer believe that this icterus is due to a polycholia, where more bile is produced than can be expelled from the gall-ducts, and that, consequently, part of it is reabsorbed. I rather consider the icterus accompanying excessive fever, as a "hæmotogene," that is, as one resulting from disintegration of the blood-corpuscles, and transformation of the released coloring matter of the blood into coloring matter of the bile. When speaking of diseases of the liver, I shall return to the subject, and will here confine myself to the following remarks. In pyæmia, in puerperal fever, and in other infectious diseases, probably as a result of the excessive increase of bodily temperature, there is often parenchymatous degeneration of the most varied organs, in which the blood also participates. More rarely, in the course of inflammatory affections, such as pneumonia, there is a *dissolutio sanguinis* (which was recognized by the older physicians), and, as a consequence of this, hæmotogenous icterus. But lastly, even catarrhal diseases, affecting the intestinal or bronchial mucous membrane, may lead to parenchymatous degeneration of the liver, heart, kidneys, or blood. During the last few years I have seen many patients with simple bronchial or intestinal catarrh die with severe nervous symptoms, icterus, moderate swelling of the liver, irregular and retarded pulse, albuminuria, etc., without there being any suspicion of an infectious disease. Such cases, which, like "bilious pneumonia," are more frequent at certain times, and occur oftener in certain regions, particularly in the tropics, doubtless form part of the bilious fevers of old writers, while another part of them were certainly cases of pyæmia and other infectious diseases.

Muriatic acid has a great reputation in the treatment of gastric fever. We are undecided as to whether the common prescription of half a drachm of concentrated muriatic acid to six ounces of mucilage, or of a weak infusion of ipecacuanha (gr. viij— ℥ vj), has the favorable effect ascribed to it. At all events, patients usually take this remedy willingly, and it moderates the thirst; and it is worthy of remark, that this prescription furnishes the gastric juice with the acid to which, as physiology shows, it owes its digestive powers.

In catarrhal fever we prescribe the alkaline carbonates, particularly the tinctura rhei aquosa. I have used this prescription, just as advised by *Schönlein*, in rather large doses, i. e., a teaspoonful every two hours, with excellent effect; and can fully support its recommendation as almost a specific in this disease. I have also observed that the patients do not well bear the customary soups, and get along better if we give them, from time to time, a small piece of black bread, sprinkled with salt.

In febris biliosa the mineral acids are usually prescribed. Perhaps an antipyretic treatment, such as quinine, in large doses, and the energetic abstraction of heat by cool baths, or repeatedly wrapping the body in wet sheets, will do still better.

# SECTION VI.

## *DISEASES OF THE PERITONÆUM.*

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## CHAPTER I.

### INFLAMMATION OF THE PERITONÆUM, PERITONITIS.

ETIOLOGY.—For the pathogeny of peritonitis we may refer to what was said of the pathogeny of pleuritis and pericarditis. The same course that we have described as occurring in the pleura and pericardium during those diseases is repeated in the peritonæum during peritonitis; while there is a new formation of young connective tissue, a proliferation in the peritonæum, its surface is covered by a fibrinous exudation, containing a variable number of young cells—pus-corpuscles. In some cases of chronic peritonitis, however, the inflammation seems to remain limited to the proliferation of the peritoneal connective tissue, and there is no free exudation. It is most probable that the thickenings and adhesions of the peritonæum, which exactly resemble those of the pleura, and, like these, are formed without symptoms, occur in this manner.

The predisposition for peritonitis, at least for the acute and diffuse form, is not great in strong, healthy persons. Slight causes, such as frequently induce inflammations of other serous and of mucous membranes, scarcely ever cause peritonitis. Hence, when a previously healthy person is attacked with peritonitis, we should suspect that it is due to one of the serious difficulties below mentioned, and should not consider it as a case of so-called rheumatic peritonitis till these other causes have been excluded, which is sometimes a difficult task. The tendency to peritonitis is much greater in persons affected with tuberculosis, morbus Brightii, and other exhausting diseases, as well as in women at the menstrual periods, than in healthy persons. Among the former, slight causes not unfrequently suffice to induce peritonitis. We have frequently given our reasons for not considering these cases of peritonitis as secondary symptoms, just as we have done the pneumonia and pleurisy which so frequently occur under the same circumstances. Finally, in not a few cases, peritonitis is the immediate result of an infection, and comes under the same category as the inflammations of the skin in exanthematous diseases. This form will be described when speaking of puerperal fever, and other infectious diseases which are "localized in the peritonæum." Among the exciting causes of peritonitis are:

1. Severe contusions and penetrating wounds of the abdomen. Among the surgical operations, paracentesis rarely leads to diffuse peritonitis, operations for hernia do so more frequently, while gastrotomy always causes it.

2. In the same way it may be caused by ruptures or perforations of organs covered by the peritonæum, and the consequent entrance of foreign bodies into the peritoneal sac. Thus perforating ulcer or cancer of the stomach, ulceration of the vermiform process, or of the cœcum, typhoid or scrofulous ulcers of the intestine, perforation of the gall or urinary bladder, opening of abscesses of the liver or spleen, etc., may cause peritonitis. In all these cases the inflammation usually spreads rapidly over the entire peritonæum. It is only rarely circumscribed by old attachments, or recent adhesions of the intestines, protecting other parts of the peritonæum from contact with the escaped substances.

3. Peritonitis may result from propagation of inflammation from other organs; it participates in the inflammation of organs covered by it, just as often as the pleura does in inflammation of the lungs. Of this nature is the peritonitis in typhlitis stercoracea, strangulated hernia, internal strangulations, rotations, and intussusceptions of the intestines. Inflammation often extends from the female sexual organs to the peritonæum. In the same way hepatitis or splenitis may cause peritonitis. In these cases the inflammation is usually circumscribed at first; and in many cases it remains so during its subsequent course; in others, particularly in those caused by incarceration and similar processes, it becomes diffuse.

4. As we have already said, peritonitis very rarely occurs in persons previously healthy, from catching cold, or from unknown atmospheric influences. When it does occur, it is called rheumatic peritonitis.

Anatomical Appearances.—We shall first speak of the appearances in *acute diffuse peritonitis.*

At the commencement of this disease the peritonæum is reddened partly by hyperæmia, partly by the escape of blood into the tissue. But, to discover this redness, it is usually necessary, first, to remove from the peritonæum the deposits which will be described hereafter.

Subsequently this redness disappears, apparently because the capillaries are compressed by the occurrence of œdema in the tissue of the peritonæum. The surface soon becomes cloudy from loss of its epithelium, and has the velvety appearance which, as we have fully described in pleuritis, depends on a proliferation of the connective tissue composing the peritonæum.

Far more noticeable than these structural changes of the peritonæum, are the exudations which never fail even after a short duration of the peritonitis. Their shape and amount vary greatly. Occasionally a thin transparent layer of coagulated fibrin, which may be peeled off like a delicate membrane, coats over the inflamed peritonæum, and unites the loops of intestine loosely together; fluid exudation is nowhere to be found. In other cases the deposit is thicker, less transparent, yellow, like croup membrane, and, in the dependent parts of the abdomen, there is a moderate amount of cloudy flocculent serum. In other cases there is a great quantity of exudation; when the abdomen is opened, an immense amount of turbid, flocculent fluid escapes, while a still greater quantity remains among the intestines, in the pelvis, and along the spine. Then, besides the membranous deposits covering the peritonæum, we find numerous yellow clumps of coagulated fibrin which partly swim in the fluid, partly sink, and collect in the dependent parts of the abdomen.

The scanty, very fibrinous exudation is chiefly found in peritonitis due to injuries or to propagation of inflammation from neighboring organs. On the contrary, the abundant sero-fibrinous exudations are more frequent in peritonitis from perforations, or dependent on infection, particularly puerperal, and lastly in the so-called rheumatic peritonitis.

All the coats of the intestines are the seat of collateral œdema, particularly in those cases accompanied by profuse exudation. Consequently the intestinal wall appears thicker; the œdema of the mucous membrane has caused serous transudation into the intestine, and the œdema and paralysis of the muscular coat have often led to enormous collections of gas in the intestine. The superficial layers of the liver, spleen, and abdominal walls, are often infiltrated and discolored. Finally, we must mention (more particularly as this partly explains the early death), that the exudation, and still more the distention of the intestines, may press the diaphragm up to the third or second rib, and compress a great part of both lungs.

If the patient does not die at the height of the inflammation, the appearances change. In the most favorable cases the fluid part of the exudation is rapidly absorbed. Subsequently the coagula and pus corpuscles, which are partly enclosed in them and partly suspended in the fluid, also disappear after they have undergone a fatty metamorphosis, become fluid, and ready for absorption; but partial thickenings and adhesions of the peritonæum always remain. In less favorable cases the absorption of the fluid part of the exudation is incomplete. The pus-corpuscles, which were at first rare in the exudation, now increase so as to give it a purulent appearance, and the fibrinous deposits also become yellower and softer. At some places the intestines adhere quite firmly and enclose the fluid, thus limiting its motions. If the patient survive this stage also, which is usually found in persons who have died in the fourth to sixth week of peritonitis, the capsulated fluid may be absorbed or thickened, and changed to a yellow cheesy or even chalky mass, which, enclosed in tough connective tissue, remains in the abdominal cavity. In other cases the extensive cell-formation occurring in the free surface of the peritonæum attacks the tissue itself, causing ulceration and perforation of the peritonæum; according to the location of this perforation, the capsulated fluid reaches the intestines or bladder, breaks through the abdominal walls, or descends into the cellular tissue of the pelvis, and escapes outwardly at some deeper point.

In *acute partial peritonitis*, the changes that we have described are limited to the serous coating of the liver, of the spleen, of a portion of intestine, or of several loops lying near together, and to the immediate vicinity of these parts. If the exudation be scanty and fibrinous, the process usually terminates with the adhesion of the inflamed parts. If the exudation be more copious and sero-fibrinous, portions may be capsulated between the inflamed parts, as in the diffuse form, and these capsulations run the course above described.

By *chronic peritonitis* is usually meant, first those cases which, beginning acutely, run a protracted course, and lead to the formation of the collections of pus above described. Secondly, those cases occurring, particularly in children, in connection with tuberculosis of the intestine and mesenteric glands, which are chronic from the start, and spread over the whole or the greater part of the peritonæum. This form is characterized by the excessive proliferation of connective tissue, as a result of which there are gelatinous or indurated thickenings of the peritonæum. The intestines usually adhere in shapeless masses, and between the various convolutions there are cavities filled with serous, purulent, or bloody fluid. The admixture of blood depends on the rupture of vessels, which usually occurs where a chronic inflammation is repeatedly lighted up, for not only the original tissue, but that which has recently formed on it, and is rich in large and thin-walled capillaries, becomes the seat of the new inflammation. Tubercles are often found in the thickened peritonæum, in this form of peritonitis; this is most apt to occur where there is hæmorrhagic exudation. Thirdly, and lastly, there is very frequently a partial chronic peritonitis, which we know better in its results than in its first stages. It occurs in chronic inflammations and degenerations of the abdominal viscera, and causes partial cloudiness and thickening of the peritonæum, adhesions of neighboring organs to each other, and distortions and folds of the intestines.

SYMPTOMS AND COURSE.—The symptoms of *acute diffuse* peritonitis at its commencement vary with the causes which induce it. Traumatic peritonitis usually begins with severe pain at the seat of the injury, which quickly spreads over the entire abdomen. In peritonitis from perforation also, excessive pain over the whole abdomen is the first symptom, if the perforation has occurred suddenly, and foreign substances have entered the peritonæum. At first, along with the pain there are symptoms of great general depression, and subsequently there is severe fever. If the perforation occurs gradually, and only a slight amount of foreign substances enters the peritonæum, the symptoms of general peritonitis are preceded by those of partial peritonitis. The commencement of an acute diffuse peritonitis, where the inflammation is propagated from neighboring organs, is far less striking. The pain already existing gradually increases; it is at first restricted to the seat of the affected organ, and thence spreads gradually over the entire abdomen. It is only in rheumatic peritonitis, and those cases resulting from infections, that we have a severe chill and intense fever at the onset of the disease, as in other severe inflammations.

No matter how the disease begins, whether there is fever at first, or it does not come on till late, pain is always the most troublesome and the most characteristic symptom. Any slight pressure on the abdomen increases it; even the pressure of the bed-clothes may become unbearable. The patient does not toss about the bed, as he does in colic, but lies on his back with the knees drawn up, and dreads every change of position. The slightest cough causes a distortion of the countenance, from pain; the patient speaks low and carefully, and does not breathe deep, fearing the pressure of the descending diaphragm. The abdomen soon becomes tense and puffed up. At first the distention depends but little on filling of the abdomen with exudation, and is mostly caused by distention of the intestines, which are filled with gas. This tympanitis is not easily explained; it is probable that it is not due to an increased formation of gas, for we can find no cause for a more rapid decomposition of the contents of the intestines; and it is just as unlikely that air should be exhaled from the wall of the intestine in peritonitis. Hence the meteorismus seems to depend, to a small degree, on the expansion of the gases due to relaxation of the intestinal walls; to a greater degree on obstructed escape of the gases due to paralysis of the muscular coat. The belly may soon become very much distended. But of course the exudation and the inflated intestines press upward against the diaphragm in the same way that they press against the abdominal walls, and so cause symptoms which, next to the pain, are the most distressing and most dangerous. The compression of the lower lobes of the lung by the upward pressure of the diaphragm, as well as the excessive hyperæmia of the non-compressed portions of lung (resulting from the disturbance of circulation in the compressed portions), induces excessive dyspnœa and a frequency of respiration of 40 to 60 inspirations in a minute. The effect of the disturbance of the circulation of the lungs may extend beyond the right side of the heart to the veins of the general circulation, and give the patient a cyanotic look. In most cases of acute diffuse peritonitis the patient is obstinately constipated; this symptom is explained by paralysis of the muscular coat of the intestines by collateral œdema. In puerperal peritonitis alone there is usually watery diarrhœa; for in this form the œdema extends to the mucous coat, and causes copious transudation into the intestines, and, if they become somewhat full, it flows away in spite of the paralysis of the muscular coat. If we set such patients up in bed, or if we press strongly on the abdomen, watery, slightly-colored masses pass from the anus. Besides the above symptoms, there is often vomiting, provided the peritonitis has not been caused by the perforation of a chronic ulcer of the stomach. At first the vomited masses are mucous and colorless, later they are more watery, greenish, or even intensely green. The causes of the vomiting, and the circumstances under which it is absent, are obscure. This difference is not explained by the participation of the covering of the stomach in the inflammation, or by its freedom from it. If the inflammation extend to the peritoneal covering of the bladder, there arises an incessant desire to urinate, and a feeling of fulness in the bladder. If an inexperienced physician be deceived by this, and be induced, by the patient's desire to urinate, to introduce a catheter, only a few drops of concentrated urine will be withdrawn. Fever is one of the symptoms of acute diffuse peritonitis, and, where this does not begin with the disease, it occurs very early. The pulse is very frequent and small; the temperature rises to 105° or more. As in any severe fever, the general state of the patient is much affected—the mind is unusually clear.

In severe cases the above symptoms become very decided in a few days. But the pain is usually worse at first, and subsequently diminishes. The belly is inflated like a drum; the liver and the point of the heart are often pressed up as high as the third rib. While at the commencement of the disease percussion gave a full tympanitic sound, after the exudation has become abundant, there is a distinct but rarely absolute dulness. The anxiety of the patient is pitiful; he beseeches aid, and looks perfectly desperate. If a quantity of blood be not abstracted, or the volume of blood be not diminished by extensive exudations, the countenance may become excessively cyanotic. Finally, the mind becomes cloudy, the patient grows apathetic and delirious, the pulse is smaller and more frequent, the body covered with cold sweat, and occasionally on the third or fourth day after the commencement of the affection, or more frequently at the end of the first week, the patient succumbs to his disease.

If the malady take a favorable course, which usually occurs only when we succeed in removing the exciting causes, or when these are not very grave, the pain, tympanites, and fever gradually subside, the respiration becomes freer, and the patient may recover rapidly. But very often, as a result of the adhesions and flexions of the intestines, habitual constipation, and occasionally colicky pains before stool, remain for life.

If the patient does not die during the first week, and if there be no decided improvement during this time, the character of the disease usually changes: it takes on a more chronic course. The pain moderates, the abdomen is only sensitive on hard pressure, the tympanitis decreases without disappearing entirely. If, up to this time, the patient has suffered from constipation, he now has movements from the bowels; if, on the contrary, there was diarrhœa as a result of the excessive transudation into the bowels, this disappears, or constipation and diarrhœa alternate. The pulse and temperature also sink somewhat, without, however, becoming normal. As the tympanites subsides, the dulness at the dependent parts of the abdomen usually becomes more distinct, and at the dull spots we perceive a gradually increasing resistance; by degrees the abdomen becomes unsymmetrical and nodular, and the capsulated exudations appear like irregular tumors. The fever, although moderated, continues and exacerbates from time to time, and it consumes not only the strength of the patient, but his blood and tissues. The fat disappears, the muscles become flabby and relaxed, the skin dry and scaly; not unfrequently there is œdema of the legs, and in the fourth, fifth, or sixth week, the patient dies of exhaustion. If, contrary to our expectation, there be reabsorption of the fluid, convalescence is very slow, and the symptoms of contraction and distortions of the intestines, which remain more constantly after these cases, are the sources of long and severe sufferings. If there be ulceration and perforation of the peritonæum, the fever increases, and, at some circumscribed spot, the abdominal walls become infiltrated, reddened, and finally the pus breaks through, or abscesses form and point at the most varied places, or, in fortunate cases, the abscesses perforate into the intestine, and the pus is passed at stool. In these cases, also, the patients usually die of exhaustion, and but few recover after tedious convalescence.

*Acute partial* peritonitis is usually preceded by premonitory symptoms, due to the disease of the organs, from which the inflammation extends to the peritonæum. Thus acute partial peritonitis, beginning in the right iliac fossa, is usually preceded by the symptoms of typhlitis; that commencing in the hypogastric, epigastric, or right hypochondriac regions, by the symptoms of ulcer of the intestines or stomach, or of abscess of the liver. The commencement of the disease itself is characterized by pain extending over the entire abdomen, but the great sensitiveness of the abdomen to pressure, which is almost characteristic of peritonitis, is limited to a circumscribed portion. Tympanites is wanting, or is, at least, partial, and the fever is more moderate than in the diffuse form. If the exudation be not extensive, these symptoms usually disappear rapidly, and the disease ends in perfect cure, unless adhesions form to disturb the movements of the intestines, or the original disease cause some other termination. When the exudation is more extensive, acute partial peritonitis runs a different course. In the vicinity of the peritonitis the percussion gradually becomes duller, the resistance of the abdominal walls more decided, till finally, in this case also, palpation shows a tumor in the abdomen. Such masses occur rarely after perforation of ulcer of the stomach; more frequently in the slow perforation of tuberculous intestinal ulcers, and in ulcerations of the cœcum, and the vermiform process. Its subsequent course is the same as that of capsulated abscesses, after protracted diffuse peritonitis.

In his clinic of abdominal diseases, *Henoch* gives a very true description of the *chronic peritonitis* which occurs, particularly in childhood, along with tuberculosis of the intestines and mesentery. He pictures the children as weak, scrofulous individuals, in whom the occasional colicky pains, the diarrhœa, alternating with constipation, and the increasing emaciation, often excite the suspicion of worms, or of tabes mesenterica. On careful examination of the abdomen, during which we must guard against mistaking the signs of displeasure for those of pain, we find it more sensitive at certain points. Sometimes even the pressure of the abdominal muscles causes pain, so that the child cries when going to stool. While the emaciation progresses rapidly, and becomes very great in a few months, while fever, toward evening, occurs regularly, the belly of the child becomes more protuberant, and gradually assumes a spherical shape. Finally, the abdominal walls become tense, even shining, and are often traversed by enlarged veins. By pressure on the abdomen, which is still painful for the child, we find an elastic resistance. The results from percussion of the abdomen vary. Only in rare cases can a free exudation be recognized by dulness in the dependent portions of the abdomen, which changes its locality with the change in position of the patient. More frequently the entire abdomen gives a dull sound, as the intestines are drawn back against the spine by the atrophying mesentery, and the exudation lies on the abdominal wall. In most cases the percussion is tympanitic at some places (where the intestines lie), and dull at others (where the fluid is). If we bear in mind this description, we shall rarely mistake this disease, which does not often occur, and which alone, or by its complications, always causes death.

*Chronic partial* peritonitis, whose remains, in the shape of thickenings, adhesions, and cicatricial contractions of the peritonæum, are found in the cadaver just as often as thickenings and adhesions of the pleura, develops just as latently as the pleuritis does, from which the pleuritic adhesions arise, and we cannot give any description of it.

Diagnosis.—Peritonitis is not readily mistaken for any other disease, as the great sensitiveness of the abdomen to the slightest pressure, the tympanites, and, in the acute form, the fever, give almost certain points for the diagnosis. Those cases dependent on perforation of ulcers of the stomach or intestines, that have not been recognized, present some difficulties of diagnosis. The sunken countenance, cool skin, small pulse, retracted abdomen, and other symptoms of severe general depression, remind us more of colic than of a severe inflammation. But if we bear in mind how insignificant the symptoms of gastric and duodenal ulcers may be, and if we observe how sensitive the abdomen is to pressure from the commencement, we shall avoid error.

On the other hand, colic, and the impaction of bilious and urinary calculi, may be erroneously considered as peritonitis; but the diagnosis is only difficult in those cases where, in hysterical women, mesenteric neuralgia is complicated with hyperæsthesia of the skin of the abdomen, in the so-called rheumatic colic, and in that from gall-stones, when the right hypochondrium is very sensitive to pressure. In these cases it may be necessary to wait for further developments before forming a diagnosis. In all other cases, the insensibility of the abdomen to pressure, or even the relief afforded by this, renders the diagnosis certain very early in the disease.

Prognosis.—Although most of the patients attacked with peritonitis die of the disease, it is not because this affection is particularly ill borne by the organism, but because it almost always depends on grave injuries or severe blood-disease, or occurs in persons previously ill, and having little power of resistance. If peritonitis be induced by the same causes on which most cases of pleuritis depend, the prognosis is unmistakably better than it is in the latter disease. Thus we frequently see the rheumatic peritonitis, which exceptionally occurs in otherwise healthy persons, particularly in menstruating women, as well as that which accompanies retention of fæces, typhlitis, or even strangulated hernia, terminate in cure, provided the exciting causes can be removed soon enough. Still less dangerous is the circumscribed chronic peritonitis, which complicates chronic inflammations and degenerations of the abdominal organs. We might regard the final object of this inflammation as an attempt of Nature to guard against future injury.

Among the symptoms on which the prognosis depends, in each case, are, in the commencement of the disease, tympanites, and the dyspnœa that it causes; the more oppressive the latter, the greater the danger. Subsequently, particularly in protracted cases, the fever and the strength and nutrition of the patient affect the prognosis more than most of the other symptoms.

TREATMENT.—Where retention of fæces, and consequent ulceration of the intestines, particularly typhlitis stercoracea, or where strangulated hernia has caused peritonitis, the *causal indication* may be answered by the treatment for the original disease and by operation, respectively. In all other cases we cannot fulfil it. This, however, is the proper place to speak of the treatment of perforation by large and repeated doses of opium; by arresting, as much as possible, the movements of the intestines, this prevents, to some extent, the contact of the escaped substances with large portions of peritonæum; and, more particularly when these foreign substances are shut off from the rest of the peritoneal cavity by adhesions, it prevents their breaking through. Statistics show favorable results for this treatment, and, in cases where, instead of opium, purely symptomatic treatment was used, and the constipation was treated by enemata and purgatives immediately after the use of these remedies, I have often seen a peritonitis, which was previously circumscribed, and might have remained so, spread over the entire peritonæum. At first we give gr. ss—j of the opium every hour, and later do not give it so often.

Concerning the indications from the disease, views have changed greatly of late. Formerly every patient treated *lege artis* was bled a pound or two; then the abdomen was covered with leeches, and one to two grains of calomel given every two hours, and at the same time a quantity of mercurial ointment was rubbed into the skin of the thighs and abdomen. "That was the proper treatment; the patients died, and no one thought of asking who recovered." It is true, we cannot claim any brilliant results from the treatment now in vogue; but that above given was just as irrational as it was injurious. On examining the bodies of persons who had died of peritonitis with abundant effusion, even when no blood had been taken, the tissues were found uncommonly bloodless, as a result of the excessive exudations. But, on examining the bodies of persons dying from a peritonitis treated *lege artis*, we find so very little blood in the heart and arteries, that we are tempted to ascribe death to the treatment rather than to the disease. If to this we add the fact that experience shows that a great loss of blood during labor proves to be no protection against an epidemic puerperal fever, and that all injurious influences which generally act as causes of peritonitis are just as active in debilitated, bloodless persons as in the strong and well-nourished, we silently pass over other reasons for avoiding venesection. (Nevertheless, we shall hereafter see that the symptomatic indications occasionally demand bleeding.) Of late scarcely any one believes in the antiphlogistic and antiplastic action of mercury, and we do not hesitate to say that we consider calomel and mercurial ointment as at least superfluous in the treatment of peritonitis, and that in purgative doses we regard calomel as directly injurious. It is far different with local blood-letting, which is much less dangerous than venesection, and of whose beneficial effect on the pain, at least, there is no doubt; this effect does not fail even in those cases where the peritonitis is caused by perforating ulcer of the stomach. The employment of cold acts in the same way, and perhaps it has even more effect on the inflammation itself. If the patient can bear it—which, unfortunately, is not always the case—we may cover the entire abdomen with cold compresses, and renew them every ten minutes. From this treatment, which is recommended by *Abercrombie*, *Kiwisch*, and others, I have seen the best results in cases that were amenable to any treatment; but I cannot deny that warm cataplasms were better borne than cold compresses by many patients. Recently the results of the opium-treatment in peritonitis, caused by perforation, and the belief that the inflamed parts needed rest more than any thing else, have rendered opium popular in the treatment of all forms of peritonitis. We agree fully with those who consider the application of leeches to the abdomen, the use of cold, and the internal administration, of opium, as the most effective treatment. If the disease be protracted, and capsulated collections of pus are formed, the persistent use of cataplasms and the early evacuation of fluctuating abscesses are urgently to be advised. The same treatment is indicated in chronic peritonitis, and the internal use of iodide of potassium, and painting the abdomen with tincture of iodine, should be tried at the same time.

In regard to the *symptomatic indications*, an early cyanosis, and still more excessive dyspnœa, if accompanied by symptoms of œdema of the lungs, requires venesection. It is true, this only temporarily removes the danger to life; but we know of no other remedy to fulfil this urgent indication. The administration of oil of turpentine, long since recommended in England, benefits the cause of the dyspnœa, the tympanites, just as little as the absorbents and other remedies, by which it has been attempted to carry gases off from the intestines. The introduction of a small trocar into the abdomen, to draw off the gas, should be avoided, as we may attain just as much by the introduction of a tube through the rectum (*Bamberger*). The vomiting is most benefited by swallowing small pieces of ice. Even the mildest eccoprotics should not be used for the constipation until the inflammation has ceased; generally, opium is as useless as the astringents in the diarrhœa depending on the œdema of the mucous membrane. In protracted cases, where there seems to be danger from the consumption caused by the fever, we should give sulphate of quinine in large doses, small quantities of wine, and a nutritious and easily-digested diet.

## CHAPTER II.

### DROPSY OF THE PERITONÆUM—ASCITES.

Etiology.—Dropsy of the peritonæum—ascites—is a transudation into the abdominal cavity, resembling the normal transudations of the body. The circumstances under which ascites develops are the same as those under which increase of the transudations occurs elsewhere, and may either be referred to increased lateral pressure in the vessels, or to a diminished amount of albumen in the blood, or, lastly, to a degeneration of the peritonæum:

1. Ascites is very often one symptom of general dropsy, whether this depend on obstruction to the flow of blood from the veins, by disease of the heart and lungs, or on degeneration of the kidneys, of the spleen, or any other disease inducing poverty of the blood. In all these cases the ascites is usually one of the last in the series of dropsical symptoms, and does not occur till dropsical effusions in the subcutaneous tissue (anasarca) of the extremities, face, etc., have existed for some time.

2. In other cases, the ascites is the result of a congestion confined to the vessels of the peritonæum. As this can only occur from an obstruction of the portal vein, it is evident that ascites occurring alone, without dropsy of any other part, accompanies diseases of the liver and its blood-vessels.

3. Lastly, ascites not unfrequently accompanies extensive degeneration of the peritonæum, such as carcinomatous or tuberculous. Of the various forms of carcinoma, however, the alveolar carcinoma of the peritonæum alone appears to be accompanied by extensive ascites.

ANATOMICAL APPEARANCES.—The amount of serum found in the abdomen varies. In some cases it is only a few pounds, in others it is forty or more. The fluid is sometimes clear, sometimes slightly cloudy, from containing cast-off and fatty epithelium. It is usually bright yellow, rich in albumen and salts, and only rarely contains flocculi of coagulated fibrin. In the fluid which is poured into the abdomen, particularly in degeneration of the peritonæum, precipitates of "late coagulating fibrin" (fibrin spätergerinnüng) form after it stands awhile in the air. This appears to depend on the fact that, in degeneration of the peritonæum, blood is often mixed with the transudations. Since *Schmidt* has shown that there is a deposit of fibrin after the addition of blood, as in all transudations, the expression "late coagulating fibrin" has acquired a different meaning.

The peritonæum itself is usually dull and whitish; the superficial layers of the liver and spleen are slightly discolored. Under the pressure of large effusions, the liver, spleen, and kidneys may become bloodless and smaller. Lastly, the diaphragm is occasionally pressed upward, to the third or second rib, by the fluid.

SYMPTOMS AND COURSE.—It is scarcely possible to give a description of ascites, as it is never an independent disease, and as its symptoms can only be artificially separated from those of the original affection.

If ascites occur during general dropsy, the subjective symptoms of the new disease, when compared with the other troubles of the patient, are usually too unimportant at first to direct attention to the ascites. Then the physical examination, induced by the suspicion that there may be ascites, gives the first certain knowledge of its existence. The case is different in the ascites accompanying disturbance of the portal circulation or degeneration of the peritonæum. When cirrhosis of the liver or cancer of the peritonæum occurs latently, the gradually increasing troubles caused by the ascites may be the first anomalies noticed, and may first excite a suspicion of the original disease. As long as the abdomen is moderately filled with fluid, the patients only complain of a feeling of fulness, and are inconvenienced by the tightness of clothes which were previously comfortable. They also notice slight difficulty on deep inspiration. If the fulness of the abdomen increase, the sensation of fulness becomes painful, and the slight difficulty of breathing increases to severe dyspnœa. The pressure of the fluid on the rectum may cause constipation, and the flatulence induced by this may increase the dyspnœa. Still more frequently the secretion of urine is diminished by the pressure of the fluid on the kidneys. It is an old belief that, after diuretics have lost their effect, they reacquire it after tapping the abdomen. This supposition apparently depends on a false interpretation of the fact that ascites adds a new difficulty to an already existing obstruction to the urinary secretion, and when it is removed the obstruction is diminished. The pressure on the vena cava and the iliac veins, where there is much effusion, causes obstruction of the circulation in the lower extremities, external genitals, and the abdominal walls. This explains the venous dilatation of those parts, and the dropsy of the subcutaneous tissue, which may become very great, and lead to mistaken ideas of the disease. We should never neglect to ask whether the legs and scrotum, or the abdomen, began to swell first.

Almost all patients with ascites are in great danger; most of them, however, do not die of the ascites, but of the original disease. The obstruction of the respiration or the excoriations and superficial gangrenes, which occasionally result from the excessive tension of the skin of the external genitals and thighs, may hasten the fatal result.

*Physical* examination of the abdomen is most important in the diagnosis of ascites. On inspection, the first thing noticed is the distention and peculiar form of the abdomen. As long as the effusion is moderate, the shape of the belly changes with every change of position of the body. If we examine the patient while he is standing, the lower part of the abdomen appears prominent; if he be lying down, the belly appears very broad. But if the transudation be excessive, the abdomen is protruded everywhere as far as the lower ribs; the false ribs themselves are elevated and pressed outwardly. Then the abdomen maintains its shape in all positions. In excessive ascites, inspection almost always shows thick networks of blue veins in the thinned coverings of the abdomen. The navel is protruded, and ruptures in the tissue of the corium form bluish-white translucent striæ, which also occur during pregnancy, if the abdomen be very much distended. If the level of the fluid rise above the brim of the pelvis, we may feel fluctuation by placing one hand flat on the abdomen and smartly tapping on the opposite side with the fingers of the other hand. Lastly, wherever the fluid is in contact with the abdominal wall, percussion is absolutely dull. At the same time it is important to notice that, except in those cases where the whole anterior surface gives a dull percussion-sound, the dulness varies with any change in position of the patient, because the fluid always goes to the most dependent part.

DIAGNOSIS.—To distinguish free dropsy from ovarian dropsy, it is imperatively necessary to obtain a perfect history of the case by a careful examination of the patient, and to pay particular attention to any possible causes of dropsy that may be discovered. The circumstances under which ovarian dropsy occurs are little known; we only know that it is often found in apparently healthy women without being complicated with any other disease. It is quite different with ascites. If we can determine that none of the anomalies of composition or distribution of the blood, described under etiology, have preceded the collection of fluid in the abdomen, and if degeneration of the peritonæum can also be excluded, in doubtful cases, the chances will be most in favor of ovarian dropsy. There are cases where the differential diagnosis depends entirely on the above factors, as the physical examination gives no decided evidence. In small ovarian cysts, it is true, the characteristic form and position of the sac, the lateral deviation of the os uteri, the similar results of percussion while the patient is in different positions, readily distinguish ovarian dropsy from ascites. But, when the cyst is very large, the peculiar form of the sac is lost; it lies in the middle of the belly, the uterus is pressed downward by the weight of the sac, but is not laterally displaced; as in extensive ascites, the percussion is dull over the entire anterior abdominal wall. *Bamberger* advises us to pay particular attention to the spot between the crest of the ilium and the twelfth rib, for, in ovarian tumors at that point, we generally find the full sound of the large intestine, in ascites we do not. Still he acknowledges that this sign occasionally fails.

After ascites has been recognized, the most important question is, what is its cause? We have already mentioned that ascites, occurring as one symptom of general dropsy, is never its first symptom. Hence, if ascites occurs in a person who has no œdema, it either depends on obstruction of the portal circulation or on degeneration of the peritonæum; it is often difficult to decide which of the two is the case. In general we may say that the coincident occurrence of symptoms of congestion in other branches of the portal vein, or the signs of disturbed action of the liver, indicate the first form; while cachexia, signs of cancer, or tuberculosis in other organs, but, above all, the presence of tumors in the abdomen, indicate the latter form. The color of the urine is very important in the differential diagnosis. For, in those diseases of the liver that lead to ascites, the urine, as a rule, contains either traces of the coloring matter of the bile or abnormal pigment; in degeneration of the peritonæum, on the contrary, it is almost always of normal color.

TREATMENT.—When the ascites is a partial symptom of general dropsy, and depends on obstructed evacuation of the vena cava, the *causal indications* require a treatment of the often-mentioned heart and lung diseases; and when it is the result of excessive hydræmia, a suitable treatment of the exhausting original disease and an improvement of the quality of blood. In the former case we are generally unable to fulfil the indications. In dropsy resulting from intermittent, morbus Brightii, and the convalescence from severe disease, the fulfilment of the latter indications usually has the best result and does much more good than the old-fashioned routine administration of hydragogues. When the portal or hepatic veins are compressed or obliterated, we cannot render them pervious again, nor can we cause an expansion of the contracting parenchyma of the liver which constricts the vessels in cirrhosis of the liver. In regard to the causal indications in ascites resulting from tuberculosis or carcinoma, we are just as powerless.

The indications from the disease demand the removal of the fluid in the abdomen. Almost all patients with ascites have diuretics prescribed them, but the number cured by these is hardly worth mentioning. If the ascites be one symptom of general dropsy, diuretics may very properly be given, but, if the result of portal obstruction, they do no more good than they would in œdema of the leg from obstruction of the crural vein by a thrombus. The case is different with drastics. Practitioners have long preferred these to diuretics in the treatment of ascites; and, in obstruction of the portal vein, we can readily see why they should be more efficient, as they cause a depletion of the branches of the portal vein, and hence diminish the increased lateral pressure, which is the cause of the ascites. In ascites, the most active among the drastics are usually chosen, and of the various compositions that have gained a reputation as hydragogues, we may mention *Heim's* pill, which, besides squills and golden sulphuret of antimony, contains chiefly gamboge. As long as the strength of the patient and the condition of his abdominal canal permit the use of drastics, they are beneficial, but if the strength fail decidedly, or the bowels become irritated, they must be given up. The operation of tapping is almost always free from danger, and it removes the fluid from the abdomen more certainly than any other method of treatment. But the more the slight danger and certain effect of tapping speak in its favor, the more necessary it becomes to enumerate its bad subsequent results. We should never forget that we do not remove water, but an albuminous fluid, from the abdomen, and that the fluid evacuated is almost always soon replaced by a new effusion. This is a severe tax on the patient's strength and supply of blood. Daily experience teaches that, after the first tapping, emaciation progresses much more rapidly than previously. From what has been said, it follows that, in ascites, the abdomen should only be tapped where life is immediately endangered by obstruction of the respiration, or by threatened gangrene of the skin.

## CHAPTER III.

### TUBERCULOSIS AND CANCER OF THE PERITONÆUM.

TUBERCULOSIS of the peritonæum hardly ever occurs primarily, but accompanies tuberculosis of either the lungs, intestines, urinary or sexual organs. In other cases it is one symptom of acute miliary tuberculosis. The latter form has no clinical interest; for the deposit of the few small, translucent nodules in the peritonæum causes no symptoms, and has no perceptible influence on the course of acute miliary tuberculosis. The few small white nodules found in the thickened serous membrane of the intestines over scrofulous ulcers are also of more pathological than clinical interest. The extensive development of the proportionately large, whitish tubercles, which are occasionally scattered through the peritonæum, is more important. The vicinity of the different nodules is either suffused with blood, or the escaped hæmatin has been changed to pigment, and the white tubercle is surrounded by a black areola. The omentum is usually rolled up, and strewn with tubercles; it forms a nodular swelling, resembling a sausage. Besides the tuberculous formations, there is usually thickening of the peritonæum from inflammatory proliferation, and there is a large quantity of fluid, sometimes bloody, in its sac.

*Cancer of the peritonæum* is also rare, as a primary disease, but is usually propagated from neighboring organs, as the liver, stomach, female sexual organs, and, more rarely, from the intestines. Scirrhus and medullary cancer usually occur as numerous granulations and nodules, scarcely so large as a pea, and scattered over the entire peritonæum, or as diffuse, flat degenerations of the peritoneal tissue. Alveolar cancer occasionally forms large, or even immense, tumors. But along with these, which are usually located in the omentum, almost all the organs of the abdomen, as well as the parietal portion of peritonæum, are also covered with small, gelatinous-looking tumors. In alveolar cancer, the intestines often adhere together in places, and the fluid in the peritoneal sac is consequently capsulated.

The symptoms accompanying tuberculosis and cancer of the intestines closely resemble those of simple ascites. The most important symptom is the gradual distention of the abdomen by the increasing collection of fluid within it. The unusual sensitiveness of the abdomen to pressure, which is absent in other forms of ascites, the rapid occurrence of cachexia, and the exclusion of all other causes for the collection of fluid in the abdomen, render it probable that there is a degeneration of the peritonæum. This suspicion can only be rendered a certainty by the discovery of one or more tumors. From the shape and extent of these tumors, from the age of the patient, and from the coincident occurrence of tuberculosis or of cancer in other organs, we decide which form of degeneration exists in the case before us.

# DISEASES OF THE LIVER AND BILE-DUCTS.

## SECTION I.

## *DISEASES OF THE LIVER.*

### CHAPTER I.

### HYPERÆMIA OF THE LIVER.

Etiology.—The amount of blood in the liver may be increased by greater afflux or by impeded efflux. The hyperæmia due to increased afflux we term *fluxion* [determination], that due to obstructed efflux, *congestion.*

*Fluxion* to the liver results—

1. From increase of the lateral pressure in the portal vein. Under normal circumstances, there is fluxion to the liver at each digestion. The passage of fluids from the intestines to the intestinal capillaries causes an increased fulness of the intestinal veins; consequently their contents are subjected to greater pressure, and are impelled more strongly toward the liver. In persons who eat and drink immoderately, this physiological fluxion becomes excessive, continues longer, is often repeated, and, like other frequently-recurring hyperæmias, may cause permanent dilatation of the vessels.

2. There is fluxion to the liver, because its capillaries, which, under normal circumstances, find a support in the parenchyma, dilate when this parenchyma becomes relaxed, and then offer an abnormally slight resistance to the blood entering the organ. The hyperæmia of the liver occurring after injuries of that viscus, or in the vicinity of inflammations and neoplasiæ, appears to develop in this way. Perhaps those cases induced by the use of spirituous liquors also belong in this class. In all of these cases we have to do with an irritation of the liver, as the alcohol is conducted directly to the liver by the portal vein; the first action of an irritant appears to consist in changes of the paren-

chyma of the irritated organ; this is usually accompanied by a diminution of resistance of the parenchyma. This diminution of resistance must result in dilatation of the capillaries, and increased flow of blood to the part. This explanation of the fact, "ubi irritatio ibi affluxus," which is so apparent in the action of warmth on the skin, is here hypothetical, it is true, but it is certainly that which best agrees with our present knowledge of the subject.

3. We are just as ignorant as to whether the cases of hyperæmia of the liver from infection of the blood with miasm, particularly with malaria, or those occurring so frequently in the tropics, depend on relaxation of the parenchyma, or whether they are due to paralysis of the muscular fibres of the efferent blood-vessels, or to a textural change of their walls, or how else they occur, as we are about the pathogeny of the hyperæmias and textural changes in the other infectious diseases. Among the cases of apparently fluxional hyperæmia of the liver, for which we can give no full explanation, are those which occur in some women just before menstruation, and are particularly marked in amenorrhœa.

*Congestion* of the liver is far more frequent than fluxion; all the blood which flows from the liver through the hepatic vein has passed through a double set of capillaries. (This is also true of the blood supplied by the hepatic artery. The capillaries formed from the hepatic artery and spreading out in the serous covering of the liver, and in its substance between the vessels and bile-ducts, unite to small venous trunks, which do not empty into the hepatic veins but into the portal veins, and, with these, again break up into capillaries.) Hence the lateral pressure in the hepatic veins is very slight. But the hepatic vein opens into the vena cava at a point where, under normal circumstances, the flow of blood meets proportionately no obstruction, as it can pour freely into the empty auricle, and particularly since during each inspiration there is a tendency of the blood toward the thorax. If there be a disturbance of these very favorable conditions for the escape of the blood, if the obstruction to its entrance from the hepatic vein into the vena cava be increased, it collects in the liver. Only a slight obstruction is necessary, for the lateral pressure in the hepatic vein is too insignificant to overcome even a very slight obstruction.

According to what we have just said, the circumstances which induce congestion of the liver are those which interfere with emptying of the right auricle. Thus it occurs—

1. In all *valvular diseases of the heart*, it appears soonest in affections of the right side, later in those of the mitral, and latest of all in those of the aortic valves. The date of the appearance of congestion of the liver in valvular disease depends, as we have previously fully

explained, on the complete or incomplete occurrence and on the longer or shorter duration of compensatory hypertrophy of the heart.

2. We may readily understand the congestions accompanying all organic diseases of the heart and pericardium, which induce obstruction to the escape of blood from the veins.

3. Under the same class would come congestions, occurring with enfeebled action of the heart, without perceptible organic change of that organ, either when appearing late in the course of exhausting acute diseases or in chronic marasmus. The effect on the distribution of the blood is the same in commencing paralysis of the heart as in degeneration of its substance.

4. Congestion of the liver is often induced by acute and chronic diseases of the lung, by which the pulmonary capillaries are atrophied or compressed, and the right side of the heart and vena cava overfilled, as in emphysema, cirrhosis, compression from pleuritic effusions, etc.

5. Lastly, in some few cases, compression of the vena cava by tumors, particularly aneurisms of the aorta, has been observed as the cause of congestion of the liver.

Anatomical Appearances.—According to the degree of the hyperæmia, the liver is more or less swollen; sometimes it is very much so; its shape is unchanged, except that it is more increased in thickness than in length. When the swelling is decided, the peritoneal coating is smooth, glistening and tense, the resistance of the liver is increased. When cut, quantities of blood flow over the cut surfaces. The latter either appear evenly dark or are spotted; this is particularly apt to occur when there has been congestion for a long time; dark spots, corresponding to the dilated venæ centrales, the commencement of the hepatic veins, and varying in shape with the direction of the cut, alternate with brighter-colored ones which do not contain so much blood, and which represent the termination of the portal vessels. The spotted appearance, which has given rise to the much misused name of *nutmeg liver*, becomes still more marked, when the more bloodless spots in the vicinity of the dilated central veins appear decidedly yellow from obstruction of the bile-ducts. The latter may be partly due to catarrh of the gall-ducts, induced by the hyperæmia of their mucous membrane; partly to the pressure of the enlarged vessels obstructing the free escape of bile from the small bile-ducts; and it may be partly due to gastroduodenal catarrh, induced by the same causes that excite the hyperæmia of the liver.

The enlarged liver may subsequently become smaller, and acquire a granular appearance, so that, on superficial examination, it may be confounded with granular liver. This is usually termed the atrophied form of nutmeg-liver. According to *Frerichs*, the atrophy and granular appearance are caused by "the venæ centrales lobulorum, and the capillaries opening into them, dilating under the strong pressure of the obstructed blood, and thus inducing atrophy of the liver-cells, lying in their network. The cells lying in the midst of the lobuli atrophy, and, in their place, appears a soft, vascular tissue, consisting of dilated capillaries, and neoplastic connective tissue." This explanation is not exactly correct, or, at least, not entirely complete. The liver cannot be diminished in size by a substitution of connective tissue, and dilated vessels for liver-cells. The diminution does not occur till the neoplastic tissue shrinks, and is reduced to a small volume. Moreover, according to *Liebermeister*, the assertion that, in congestive hyperæmias, the proliferation of connective tissue occurs particularly in the vicinity of the venæ centrales, is based on theoretical grounds, and not on direct observation. On the contrary, *Liebermeister* found that, in the atrophic form of nutmeg-liver, as well as in cirrhosis, the proliferation affected chiefly the vicinity of the venæ interlobulares, and in some cases led to a typical development of interlobular tissue, which, as is well known, hardly exists in healthy human livers.

Symptoms and Course.—There are neither subjective nor objective symptoms of the disease until the hyperæmia of the liver has attained a high grade, and the organ has considerably increased in size. Where the liver is decidedly enlarged, the patients feel that their right hypochondrium is unusually full, and this sensation of fulness not unfrequently increases to a painful feeling of tension, which spreads from the right hypochondrium over the abdomen. The pressure in the right hypochondrium, or the sensation of having a firm hoop around the abdomen is often, next to the dyspnœa, the chief complaint of patients with heart-disease. And when the liver swells, the sufferings of emphysematous patients, and of those affected with cirrhosis of the lungs, or curvature of the spine, are decidedly increased. Patients with extensive hyperæmic swelling of the liver cannot bear tight clothes, as they interfere with deep inspiration. If, from the causes above mentioned, a slight obstruction of the gall-ducts accompany the hyperæmia of the liver, there will be some icterus; and, as the patient's color is already somewhat bluish (cyanotic), from the obstruction of the venous circulation, he will have the peculiar greenish color characteristic of patients with heart-disease shortly before death. Besides the symptoms just mentioned, and the physical observation that the liver is enlarged, in simple hyperæmia there may be no symptoms of disturbed hepatic function. A slight increase or diminution of the secretion of bile may escape our observation during life; even in the cadaver, where there was excessive congestive hyperæmia, *Frerichs* could not make out such a change. In some few cases only, did he find the bile albuminous. Patients with hyperæmia of the liver have other complaints, it is true; they suffer from headache, difficulty of digestion, irregularity of the bowels, hemorrhoids, etc. These troubles, however, are not the result of the hepatic engorgement, but may have no connection with it, or, as is more frequently the case, they depend on the same causes. Diseases of the heart not only induce hyperæmia of the liver, but also lead to gastric and intestinal catarrh; in the same way, excess in eating and drinking excites gastric and intestinal catarrh even sooner than it does hyperæmia of the liver. It appears to be different with those cases of hyperæmia of the liver which frequently occur in the tropics, probably from malaria. These begin with great constitutional disturbance, severe headache, bilious evacuations upward and downward, and often with the passage of bloody mucous masses. These symptoms of this disease—which is but little known—decidedly favor the idea that it is not a simple hyperæmia, but either a coincident anomaly of secretion of the liver, independent of the hyperæmia, or the first stage of a severe organic disease, which, in fact, not unfrequently develops more fully. But perhaps in these cases, also, the hyperæmia of the liver is only the partial expression of a disease affecting all the abdominal organs, particularly the intestines; and this view would best explain the constitutional affection, and the other symptoms.

When the hyperæmia has reached a high grade, physical examination very clearly shows the swelling of the liver. As we now, for the first time, speak of the physical signs of enlargement of the liver, we must give some account of them.

As physical aids for recognizing enlargement of the liver, we have inspection, palpation, and percussion.

In decided swelling of the liver, *inspection* shows a prominence in the right hypochondrium, extending more or less toward the left side, and gradually disappearing inferiorly. At the same time, the right side of the thorax, which even normally is from half an inch to an inch larger than the left, becomes more prominent at its lower part. Lastly, the inferior ribs may be elevated by the enlarged liver, pressed close together, and their lower edges turned forward.

If we do not undertake the examination very quietly and carefully, the contractions of the abdominal muscles, which usually occur, greatly interfere with *palpation*. The inexperienced often mistake contracted portions of the rectus abdominis for tumors of the liver. We should never undertake the examination while the patient is standing or sitting. He should lie down, and draw up the knees a little. At the same time, we should tell him to respire regularly, and should distract his attention from the examination, by questions, etc. In many cases of enlargement of the liver, that can be certainly recognized by percussion, it is true, we find great resistance in the right hypochondrium, but we cannot clearly make out the edge of the liver. This is particularly the case where the resistance of the liver is not increased, and still more so when it is diminished. In other cases, palpation gives the best evidence concerning the amount of enlargement, and also, about the shape of the margin and surface; and this is the more distinct the greater the resistance of the enlarged organ.

*Percussion* is the most important physical aid in the diagnosis of the enlargement of the liver. In determining the upper boundary, it is not customary to decide by the commencing flatness of percussion at those points where there is a thin layer of lung between the liver and the walls of the thorax, but by the absolute dulness where the liver comes in contact with the thoracic wall. Hereafter, in speaking of the upper border of the liver, we shall always mean the line of absolute dulness. The highest point of the liver lies about 3 cm., above this line. Normally, on the mammillary line, the upper margin of the liver lies at the lower border of the sixth rib; on deep inspiration it descends to the seventh rib, on complete expiration it ascends to the fifth. In the axillary line the upper margin lies about the eighth rib, near the spine about the level of the eleventh rib. In the median line, the upper margin of the liver lies on a level with the union of the xiphoid cartilage and the body of the sternum, but its position cannot usually be determined, as the liver dulness passes into that of the heart. Normally, in the mammillary line, the lower border of the liver lies at the margin of the ribs or a little below; in the axillary line it is usually above the eleventh rib; in the median line about half way between the xiphoid cartilage and the navel; near the spine its position cannot be determined. As the thorax is shorter in women and children, the lower border of the liver lies somewhat below the edge of the ribs. Neither the sharp border of the liver, which extends a few centimeters below the ribs, nor its left lobe, if not thickened, causes any decided dulness on percussion. In forty-nine cases, of persons between twenty and forty years of age, examined by *Frerichs*, the distance from the upper line of dulness to the lower averaged, in the mammillary line, 9.5 cm., in the axillary line, 9.36 cm., in the sternal line, 5.82 cm. (The observations of *Bamberger* differ decidedly from these: in thirty measurements made in adults, he found in the mammillary line, in women, 9 cm., in men, 11 cm., in the axillary line, in women, 10.5 cm., in men, 12 cm., and in a line one inch to the right of the median line, in women, 8.5 cm., in men, 11 cm., as the average extension of dulness.)

If the liver be enlarged, the liver dulness will extend into the right hypochondrium and epigastrium. Near the edge of the liver the dulness becomes indistinct or disappears entirely, a fact which we must know, or we shall suppose the organ is smaller than it really is. Before deciding, from the extension of dulness into the right hypochondrium, that the liver is enlarged, we must determine that it is not displaced downward. We have already spoken at length of some important points for the diagnosis between enlargement and displacement of the liver. Moreover, without being enlarged, a greater part of the liver may lie in contact with the abdominal walls, if it has sunk downward from pressure on the lower part of the thorax or from relaxation of its tissue, or if it has an abnormal form. Among the anomalies of form the most frequent are those induced in women by tight-lacing and still more by wearing their waistbands tight. As a result of this constant pressure on it, without increase of its volume, the liver may become much flattened and so elongated as to descend several finger-breadths below the ribs, or in some few cases even down to the crest of the ilium. We must bear in mind these deviations in position and form of the liver, if we would rightly interpret the results of physical examination.

Hyperæmic swelling of the liver can rarely be perceived by simple inspection. From the decided increase of thickness of the organ, percussion gives great dulness, which may extend from the right to the left hypochondrium, and as far down as the navel or even below it. And as the resistance of the liver is increased, we can usually feel its margin, and satisfy ourselves that its form is unchanged and its surface smooth. A characteristic of hepatic enlargements caused by hyperæmia is that they grow more rapidly than any other form, and decrease again rapidly.

It is remarkable that the symptoms of atrophic nutmeg-liver have not received proper attention until lately, when *Liebermeister* has paid a great attention to them. As the symptoms of this disease are characteristic, it may be quickly sketched. The patients affected have disease of the heart or emphysema, or some other disease of the lung, which impedes the flow of blood through the right side of the heart. This obstruction of the circulation has caused enlargement of the liver, cyanosis, and general dropsy. As is usual in heart and lung diseases, the dropsy began in the lower extremities, and afterward invaded the serous cavities. Subsequently the state of affairs is changed: the ascites becomes more decided than the anasarca; or the ascites continues, while we may succeed in removing the other symptoms of dropsy for a time by suitable remedies. On examining the liver, we find it smaller than at first, its lower border is some finger-breadths higher than it was some weeks or months previously. Not unfrequently, while the anasarca is moderate, the ascites increases, so that we are compelled to tap the patient. In the cases that I have observed, where in heart and lung diseases I have been able to diagnose atrophic nutmeg-liver from the disproportion between the ascites and anasarca and from the perceptible diminution in size of the enlarged liver, I have not found the spleen enlarged. If we rightly understand atrophic nutmeg-liver, the explanation of the above symptoms is not difficult. The contraction of the connective tissue of the liver compresses the vessels, hence the flow of blood from the veins of the peritonæum is opposed in two ways: first, by the heart or lung affection; secondly, by the compression of the hepatic vessels. The absence of enlargement of the spleen is the only thing that could appear remarkable; as, by compression of the vessels of the liver, the flow of blood from the spleen is also obstructed, and since in cirrhosis of the liver (see Chapter III.), where the same circumstances prevail, we almost always find enlargement of the spleen, and usually ascribe this enlargement to obstruction of the blood in the splenic vein. I propose, when speaking of cirrhosis of the liver and hyperæmia of the spleen, to discuss this apparent contradiction more fully.

Treatment.—The causal indications require the removal of the circumstances inducing the fluxion to or the congestion of the liver. In the fluxions caused by excess in eating and drinking, the diet is to be regulated; in those cases resulting from misuse of spirituous liquors, alcohol should be forbidden. In the same way it may be necessary to advise a change of residence when persons in the tropics, or from the influence of malaria, suffer repeatedly from hyperæmia of the liver. Finally, if severe fluxions to the liver occur just before the menses or during their absence at the time they are expected, the causal indications require the application of leeches to the os uteri or of cups to the inner surface of the thighs. In congestion of the liver we are either unable to fulfil the *indicatio causalis* or, where we can do so, it is almost always some other trouble than the hyperæmia of the liver, which decides us to interfere. For instance, when we bleed in pneumonia, and thus moderate a congestion of the liver, the venesection has not been induced by the latter, but by the congestion in the brain, or some other cause.

For the fulfilment of the *indications from the disease*, the abstraction of blood from the region of the liver, which is so frequently recommended, is just as irrational as it is inefficacious, and *Henoch* is right in saying that the leeches would do just as much good if applied to the wrist or ankle-joints as when applied to the right hypochondrium. On the contrary, leeches about the anus are strongly to be recommended, if the sufferings of the patient render it worth while to use them at all. They draw blood from the anastomoses of the branches of the portal vein, and thus lessen the lateral pressure in the portal vein, and consequently the supply of blood to the liver. Laxatives have a similar influence, particularly the neutral salts, as, by abstracting water, they also cause depletion of the intestinal veins, and thus diminish the lateral pressure in the portal veins. For patients who habitually suffer from hyperæmia of the liver, the mineral springs at Homburg, Kissengen, Marienbad, etc., are particularly beneficial, for the salts, in the form in which they there enter the body, can undoubtedly be used for a long time without injury.

## *INFLAMMATIONS OF THE LIVER.*

For sake of convenience, we shall make a general division of five forms of inflammation of the liver, of each of which we shall hereafter give a more accurate description: 1. Suppurative hepatitis; 2. Chronic interstitial hepatitis, which, in its later stages is called cirrhosis hepatis; 3. Syphilitic hepatitis; 4. Pylephlebitis; and 5. Acute yellow atrophy of the liver, which is reckoned among the inflammations of the liver, at least by most recent pathologists. We shall not treat of the last form till we have considered other affections of the liver, accompanied by jaundice, that are more readily understood.

## CHAPTER II.

### SUPPURATIVE HEPATITIS.

Etiology.—According to *Virchow*, the processes observed in this form of hepatitis originally affect the liver-cells themselves. At first these swell from imbibition of an albuminous substance; subsequently there is disintegration of the cells, and consequently of the parenchyma of the liver; finally, there are cavities in the liver which are filled with the disintegrated elements of the tissue. On the other hand, *Liebermeister* thinks that his examinations prove that, in suppurative hepatitis, the process starts from the interstitial tissue; and that the disintegration of the liver-cells is secondary. The etiology of parenchymatous hepatitis is obscure. It is rare in the temperate zones, but more frequent in the tropics, particularly in India, although the old accounts of its frequency there are overdrawn.

Among the exciting causes, we may mention—

1. Wounds and contusions of the liver; but among sixty cases that he observed himself, or collected from other observers, *Budd* found only one case due to injury.

2. Very similar to the above are the cases induced by impaction of angular concrements in the gall-ducts, but these must be considered as very rare.

3. Suppurative hepatitis develops more frequently from ulceration or other gangrenous affections of the abdominal organs. It has been found complicating ulcers of the stomach, intestines, and gall-bladder, and, in some few cases, abscesses of the liver have been seen to follow operations for hernia or about the rectum. In these cases it is most natural to suspect an embolus of the branches of the portal vein or the transfer of injurious irritating matter to the liver by the portal blood; but, so far, it has been impossible to obtain any positive proof of this. *Budd* believes, and most authors agree with him, that the majority of cases of hepatitis of the tropics belong in this class. It is true that it is very rarely a primary disease, but is almost always secondary to the dysentery endemic in the tropics; but it has not been proved in this form either, that the transfer of particles of gangrenous mucous membrane or of putrid fluid, from the large intestine to the liver, has induced the inflammation in the latter, and still less that this is the sole cause of hepatitis in the tropics. The fact that epidemic dysentery in our country is hardly ever complicated by hepatitis, although with us also there are extensive gangrene of the mucous membrane and putrid decomposition of the contents of the large intestine, rather militates against *Budd's* view of the subject.

4. Lastly, among the exciting causes of parenchymatous hepatitis, we must mention injuries, suppurations, thromboses, and inflammations of the veins. The explanation of this form, which, together with the above, is usually called metastatic hepatitis, is very difficult. We have given the views at present prevalent concerning the formation of metastases in the lungs. According to the explanations there given, the occurrence of metastases in the liver from peripheral suppurations should induce the belief that emboli which have passed through the capillaries of the lungs may plug up the hepatic artery. We must content ourselves with having mentioned the fact, and called attention to the difficulty of its explanation. The sympathy between the head and the liver, of which we have already spoken a good deal, can only be explained by the fact that wounds of the skull affecting the diphœ very readily induce metastases, and, under some circumstances, metastases to the liver.

ANATOMICAL APPEARANCES.—In parenchymatous hepatitis, the entire organ is never inflamed, but there are always inflamed spots.

These vary in size; often there is only one; in other cases, numbers of them are scattered through the liver.

We rarely see the *post-mortem* appearances of the disease in the commencing stage. The description of the inflamed parts as dark-red, resistant places, which become slightly prominent when we cut into the engorged liver, is probably taken more from analogy than from actual observation. On the other hand, in commencing hepatitis, we do find discolored, yellowish, and very soft spots in the hyperæmic liver. When these are superficial, before opening them, we may readily mistake them for abscesses. At these spots, on microscopical examination, *Virchow* found, according to the degree of discoloration and softening, either that the liver-cells were cloudy, transparent, and granular, or that their number was decreased, and between those still existing there was effused a finely-granular mass, or, lastly (at the most discolored and softened spots), that the liver-cells had entirely disappeared, and in their place there was only a finely-granular detritus.

Far more frequently, parenchymatous hepatitis is not seen till its later stages. Then we find abscesses in the liver, from the size of a pea to that of a hen's egg; if several of these have united, or if the disintegration has progressed further, these form irregular collections of pus, which are often very large. They are surrounded by disintegrating, discolored parenchymatous substance, and contain a creamy pus, which is often greenish from admixture of bile. When the destruction advances to the surface, these abscesses of the liver may break. This may occur into the abdominal cavity, or, if there has been previous adhesion of the liver to the abdominal wall, the perforation may be outwardly; in other cases, after the liver has become adherent to the diaphragm, the latter is perforated, and the pus enters the pleural sac or the lung, if that be adherent to the pleura costarum. In rare cases, abscesses of the liver have been known to perforate into the pericardium, stomach, intestines, gall-bladder, even into the portal vein, and into the ascending vena cava.

If the patient lives after the opening of the abscess, in favorable cases, the walls may unite; then there is a proliferation of connective tissue, and finally a hard cicatrix forms, which often contains thickened and calcified masses of pus. And where perforation does not occur, after an abscess has existed a long time, there is usually a proliferation of connective tissue in the wall and vicinity of the abscess; its inner surface becomes smooth, the pus is incapsulated, and gradually thickened by reabsorption of its fluid constituents. Then the abscess may be diminished in size by the shrinking of the surrounding connective tissue, till finally only a dense cicatrix remains, enclosing a calcareous mass.

SYMPTOMS AND COURSE.—The typical description usually given of parenchymatous hepatitis answers, as *Budd* aptly says, only for the traumatic cases, including those induced by impacted gall-stones; but, as we have already stated, this is the rarest form of the disease. If, after a blow, or some other violence, affecting the region of the liver, there be severe pain there, if the liver swells, and there be high fever, with general suffering, there is no difficulty in the diagnosis.

The case is altogether different when hepatitis comes on during some gangrenous process in the abdomen, when it complicates a dysentery, or when it develops during peripheral suppuration, after injuries of the head, great surgical operations, etc. The cases related by *Budd*, *Andral*, and others, show a number of instances where abscesses of the liver, occurring in this way, were recognized very late, or not at all. In chronic ulcerations of the intestines, in perityphlitis, and similar diseases, as well as after operations on the rectum or abdomen, we may suspect the occurrence of hepatitis, if the patient has a chill, if the liver swells and becomes painful, and there be icterus. But none of these symptoms are constant; and cases where local symptoms of hepatic disease are absent, in metastases starting from one of the abdominal organs, are at least as frequent as those of metastases in the lungs, which run their course without pain in the chest or bloody expectoration. The chills and fever may also depend on other causes, and cannot, by any means, be regarded as sure signs of secondary hepatitis. It is still more difficult to recognize the occurrence of hepatitis during endemic dysentery. For, in this disease, the liver is not unfrequently swollen and painful, even when it is not inflamed; the fever present does not decide the question, for it is present in dysentery alone; in many cases there is no icterus, and, when it occurs, it is no sure sign of hepatitis. The parenchymatous hepatitis which occurs in peripheral suppurations, or after surgical operations, and is one symptom of the so-called pyæmia, is the most difficult to recognize. Under such circumstances, we should not expect the prostrate patient, who is much depressed mentally, to complain of pain about the liver; the chills and high fever, and even excessive icterus, do not render it absolutely certain that there is disease of the liver.

If we add, to what has already been said, that the abscesses, which have formed during the above-named diseases, always enlarge slowly, and are accompanied by very unimportant symptoms, it may readily be understood that a subsequent chronic disease, with the symptoms hereafter mentioned, may first excite the suspicion, or render it certain that the original disease has been complicated with hepatitis.

The symptoms of the abscess which remains and gradually increases are quite varied. There is almost always a dull pain in the right hypochondrium, which is increased by pressure. Occasionally there is also a peculiar "sympathetic" pain in the right shoulder, whose frequency and diagnostic importance were formerly much overrated. The liver almost always projects below the ribs, and in cases where the abscesses are large or numerous, or the hyperæmia is great, the liver may be double its normal size, and bulge out the right half of the thorax, render the hypochondrium prominent, and project deep into the abdomen. When the abscesses are on the convex surface of the liver and are somewhat prominent, we may sometimes, on careful palpation, find slight protuberances or even fluctuation. Icterus is not at all a constant symptom of abscess of the liver, being absent even in the majority of cases. The accumulation and absorption of bile, on which icterus depends, are partly the result of compression of the gall-ducts, and partly due to their obstruction by albuminous and fibrinous coagula (*Rokitansky*). Large abscesses may compress the ramifications of the portal vein; such as project from the concave surface may compress its trunk. In such cases, besides the symptoms above described, there are usually swelling of the spleen and serous effusion into the abdomen. While the abscesses are small there is little or no accompanying fever, and at this time the general health of the patient is little affected, his strength is good, and he may live for years in passable health. But as soon as the abscess has attained some size the fever becomes higher, chills come on from time to time, as we have seen that they do in chronic suppurations elsewhere, the strength and nutrition of the patient suffer, he becomes cachectic and excessively emaciated, and in most cases finally dies exhausted and dropsical, of "consumption of the liver."

If the abscess of the liver perforate into the abdomen, the symptoms of peritonitis soon set in and quickly cause the death of the patient. If the abscess become adherent to the anterior abdominal wall, this at first becomes œdematous and finally infiltrated; this renders any formerly perceptible fluctuation indistinct, but a superficial fluctuation gradually occurs in the abdominal wall, and this is finally perforated by the pus. If the perforation take place through the diaphragm, we either have the symptoms of pleurisy, or, more frequently (as the pleural surfaces have become adherent), dark-red or brown purulent masses are suddenly thrown off, from whose appearance *Budd* claims to have frequently made a diagnosis of abscess of the liver. From perforation into the pericardium, pericarditis rapidly develops and soon causes death. In perforation of the stomach, the peculiarly colored masses are vomited. In perforation of the intestine, on the other hand, there are purulent passages from the bowels.

When the pus is evacuated outwardly or into the stomach, or intestines, or even when it enters the bronchi and is coughed up, the patients usually feel instantaneously relieved; the improvement rarely remains permanent, however, and, when it does so, it is only in cases where the abscess was small. *Budd* only saw closure of the abscess and perfect cure of the patient in one case after the evacuation of the pus. The abscess usually continues to suppurate, and the patients sooner or later die of exhaustion from the suppuration and fever. Cases of cure after capsulation and gradual decrease in size of the abscess, with inspissation of its contents, are very rare, and during life it must be difficult to decide that this has occurred.

TREATMENT.—It is only in the rare cases of traumatic hepatitis that we can hope to induce resolution of the inflammation by the use of cold compresses, and the application of leeches about the anus. In the subsequent course of the disease, blisters over the liver, and the internal administration of calomel, are very generally employed, but are of doubtful efficacy.

In all other forms of parenchymatous hepatitis, we have to confine ourselves to the treatment of symptoms, particularly as they are rarely recognized until abscesses have formed. Fortunately for the patient, the views on which it was formerly maintained, that the reabsorption of pus was aided by the internal and external use of mercurials, are no longer held; although it is said that patients with liver-disease are just the ones who can take large doses of calomel without injury.

As long as there is no perceptible fluctuation, and we cannot open the abscess, we must limit ourselves to keeping up the strength of the patient by suitable diet, wine, and preparations of iron. For the chills we prescribe quinine, which not unfrequently has a very decided antiperiodical action.

As experience shows that those abscesses heal best from which pus mixed with blood and broken-down parenchyma of the liver have been evacuated, while those containing good or laudable pus rarely healed, we should make it a rule to open the abscesses as early as possible, before a so-called pyogenic membrane has formed in them. Surgery teaches us to be peculiarly careful in opening abscesses of the liver, and that we should use caustics instead of the knife, where we cannot certainly determine that the liver has become adherent to the abdominal walls.

## CHAPTER III.

### CHRONIC INTERSTITIAL HEPATITIS, CIRRHOSIS OF THE LIVER, GRANULAR LIVER.

ETIOLOGY.—Interstitial hepatitis affects the fibrous covering of the liver and the scanty connective tissue, which, as the continuation of

*Glisson's* capsule, accompanies the hepatic vessels and traverses the parenchyma of the liver. In this form of inflammation there is neither free exudation, suppuration, nor formation of abscesses. The inflammatory process rather consists in a proliferation of the tissue above named, by the formation of young connective-tissue elements from those already existing. While the connective tissue of the liver increases, its parenchyma proper is more and more displaced. In the later stages of the disease the neoplastic tissue undergoes a cicatricial retraction, which strangulates and partly destroys the parenchyma of the liver. The blood-vessels and bile-ducts not unfrequently become impervious throughout a considerable extent, and a large part of the liver-cells atrophy and die.

Alcohol is the irritant which most frequently induces interstitial hepatitis. Hence English physicians give granular liver the vulgar name of "gin-drinker's liver." Corresponding to the extent to which alcohol is used by the sexes and at different ages, the disease is more frequent in men than in women, and is very rarely seen during childhood. Even the apparent exceptions support the rule. Thus *Wunderlich* found typical cases of the disease in two sisters aged eleven and twelve years; but on careful inquiry it was found that both of them were great schnapps-drinkers.

The use of alcohol, however, is not the only cause of interstitial hepatitis, and all persons affected with this disease, who deny the habitual use of liquor, are not to be regarded as secret topers. Simple congestive hyperæmia, which so often occurs in heart-disease, has frequently been blamed as a cause of cirrhosis; but, according to *Bamberger's* numerous observations, this is probably an error, which was due to the atrophic form of nutmeg-liver being confounded with cirrhosis. We are unacquainted with the other causes of interstitial hepatitis. *Budd* (as quoted by *Bamberger* and *Henoch*) says: "There may be other substances, among the immense variety of matters taken into the stomach, or among the products of faulty digestion, which, on being absorbed into the portal blood, cause, like alcohol, adhesive inflammation of the liver." *Budd* himself, however, terms this view hypothetical. I have seen one case where biliary calculi caused chronic interstitial hepatitis. In this case, which was fully detailed by *Liebermeister*, most of the large bile-ducts were filled with stony concrements, and the liver was most strikingly cirrhotic in character.

Anatomical Appearances.—In the first stage, which we rarely see, the liver is increased in size, particularly in thickness; its peritoneal covering is slightly thickened and clouded; except some slight elevations, the surface is smooth and even. On section, the parenchyma is seen to be interspersed by a vascular, succulent, grayish-red mass, which gives the liver a fleshy look, and which is shown by the microscope to consist of delicate connective-tissue striæ with spindle-shaped cells. Between this tissue, the original parenchyma appears as large and only slightly-prominent granulations.

The *second* stage, into which the first gradually passes, is excellently described by *Rokitansky*. According to him, in typical cases, the liver is much smaller than normal; its form is altered, the edges becoming thinned, and finally changed to an indurated border, which contains no liver-tissue: on the other hand, the thickness, particularly of the right lobe, is relatively greater. Lastly, the whole organ often consists of the spherical right lobe, to which the left is attached as a flat appendix. In this stage we may see granular or watery projections (granulations) on the surface, to which the disease owes its name of "granular liver." If the granulations are all of the same size, as large as a hemp-seed, for instance, the surface appears regularly granular; if they vary in size, it is irregularly granular. Between the prominences the serous coat is whitish, tendinous, shrunken, and retracted; if deep retractions separate large portions of the liver from each other, it appears lobular. The serous coat is also usually attached to the surrounding parts, particularly to the diaphragm, by short, firm adhesions, or by bands. The substance of the cirrhotic liver is very hard and of leathery toughness. On section, there is often as much resistance as on cutting into scirrhus, and on the cut surface we find the same granulations as on the surface of the liver. They are embedded in a dirty-white, dense, non-vascular tissue. At some places the parenchyma has entirely disappeared, and the dense tissue alone remains. On microscopic examination in this stage, we no longer find the first elements of connective tissue, but this is fully formed, and encloses in concentric layers groups of liver-cells (the granulations). The still existing liver-cells are partly affected with fatty degeneration and partly intensely yellow, as a result of the retention of bile induced by obstruction of the bile-ducts. The fatty metamorphosis of the liver-cells, and still more the pigment in them, gives the entire liver, but particularly the granulations, the yellow color to which it gives its name, "cirrhosis."

SYMPTOMS AND COURSE.—The symptoms of the first stage of cirrhosis are very similar to those of simple hyperæmia of the liver; the inflammatory process within the liver and in its covering is usually accompanied by little pain, although the patient may be more sensitive to pressure over the liver than is the case in simple hyperæmia. In some cases, however, the feeling of fulness in the right hypochondrium increases to painful tension or even to burning pain. Besides these symptoms, there are various troubles in the first stage of cirrhosis.

The patients complain of loss of appetite, of a feeling of pressure and fulness after eating; they suffer from flatulence and constipation. The nutrition may be already affected and the appearance cachectic; but the same is true of these appearances as has been said of several of the accompaniments of simple hyperæmia of the liver, that, although they accompany the disease, they are not symptoms of it. The habitual use of liquor almost always causes chronic catarrh of the stomach, and the symptoms depend on this, not on the interstitial hepatitis.

The symptoms of the second stage depend almost entirely on mechanical conditions. Compression of the branches of the portal vein must cause symptoms of congestion in those organs from which the portal vein conducts the blood to the liver; the compression of the bile-ducts (as long as the liver-cells to which they belong prepare bile) induces absorption of bile and icterus.

Symptoms of congestion are seen soonest and most frequently in the gastric and intestinal mucous membrane. The chronic gastric catarrh accompanying the second stage of cirrhosis is not, as in the first stage, a complication, but is a necessary result of the disease. The symptoms it causes have already been described. Intestinal catarrh, which is just as frequent an accompaniment of cirrhosis, rarely leads to excessive transudations of fluid into the intestines, but, like most chronic catarrhs, to a copious production of cells and to the secretion of tough mucus. We have learned that constipation, tympanites, cachectic appearance, etc., are among the symptoms of this form of chronic intestinal catarrh, hence we readily understand why they should take a prominent part among the symptoms of cirrhosis of the liver. Not unfrequently the capillaries of the gastric and intestinal mucous membrane become so full as to rupture. Hence, next to ulcer of the stomach, cirrhosis of the liver is the most frequent cause of gastric and intestinal hæmorrhages; and as the obstructed evacuation of the portal vein causes overfilling of the inferior mesenteric artery and the hæmorrhoidal plexus, it is to be mentioned among the causes of hæmorrhoids, and these form one of the most frequent symptoms of cirrhosis.

As the splenic vein also empties into the portal vein, compression of the branches of the latter will impede the escape of blood from the former; hence symptoms of congestion of the spleen unite with those of the gastric and intestinal congestion. In the later stages of interstitial hepatitis the spleen has so often been found enlarged to two or three times its natural size, or even more, that *Oppolzer*, *Bamberger*, and others, give enlargement of the spleen as one of the most important symptoms of cirrhosis of the liver. Out of thirty-six cases, *Frerichs* found the spleen enlarged eighteen times. We cannot agree, however, in referring the enlargement of the spleen solely to obstruction of the blood: on the one hand, because it sometimes comes very early, at others very late, without any corresponding change in the other symptoms of congestion; secondly, because in some cases of cirrhosis, and in all the cases of atrophic nutmeg-liver that we have observed, in spite of the excessive compression of the hepatic vessels, there has been no enlargement of the spleen, or it has been only slightly enlarged. Probably the swelling of the spleen depends, partly at least, on a process similar to that affecting the liver. But, that part of the splenic enlargement is due to obstruction of the flow of blood, is shown by the constant diminution in size of the spleen, when a hæmatemesis, from rupture of the capillaries of the stomach, has facilitated the escape of blood from the spleen.

As the veins of the peritonæum also, particularly those of its visceral folds, empty into the portal vein, we may readily understand the occurrence of ascites, which is the most apparent symptom of cirrhosis. We may refer to the chapter next to the last of the previous section, where the increased lateral pressure in the veins of the peritonæum was shown to be the most important cause of serous transudations into the abdomen. As rupture of the capillaries of the peritonæum occasionally occurs, in some cases, we find small quantities of blood mixed with the transudation. In other cases, there are flocculi of fibrin in the fluid, which tend to prove that, while the inflammation is going on in the liver and its vicinity, small quantities of free exudation are formed. The ascites, which forms a symptom of cirrhosis of the liver, is particularly extensive; hence in it, more frequently than in any other form of abdominal dropsy, we find the blue veins over the abdomen, œdema of the lower extremities, genital organs, and abdominal walls, resulting from compression of the vena cava and iliac veins, as well as the superficial gangrene of these parts that we have previously described.

Now that we have mentioned chronic gastric and intestinal catarrh, gastric and intestinal hæmorrhages, hæmorrhoids, enlargement of the spleen, and, lastly, ascites, as the almost constant symptoms of cirrhosis, and as the mechanical results of compression of the branches of the portal vein, it may be asked how we explain the exceptions, where these symptoms do not exist, or are insignificant. We shall first remark that, occasionally, in spite of advanced cirrhosis, the branches of the portal vein remain quite pervious, so that, according to the observations of *Förster*, in some cases, they may be traced a considerable distance in the cadaver. But, besides this, the escape of blood from the stomach, intestines, spleen, and peritonæum may be facilitated, and the congestion in these organs avoided, by the blood seeking other passages, and the development of a collateral circulation. This may result: 1. From the connection between the inferior mesenteric and the hypogastric veins, through the hæmorrhoidal plexus; 2. From the anastomoses between the portal veins and those veins of the peritonæum which open into the diaphragmatic and œsophageal veins; 3. Through newly-formed vessels in the adhesions between the liver and diaphragm. Besides these ways, and other occasional abnormal communications, by which the blood from the portal vein may elude the hepatic vein and reach the vena cava, in some cases, 4, a very peculiar form of collateral circulation is set up, which may be recognized, even during life, by very evident symptoms. It was formerly supposed that this form only occurred when the umbilical vein was incompletely closed after birth, and that a fine canal remained in the ligamentum teres during after-life. If considerable congestion of the liver occur in such cases, this fine canal is gradually distended by the pressure of the blood, and may become so pervious as to conduct the blood to the anterior abdominal wall, where it empties into the ramifications of the internal mammary veins. The consequent overfilling of the internal mammary veins impedes the escape of blood from the cutaneous veins, so that these may be excessively dilated, and surround the navel as a blue cushion. The deformity thus induced, the *caput Medusæ*, does not, however, depend on dilatation of the incompletely obliterated umbilical vein, but on dilatation of the branches of the portal vein running from the liver to the anterior abdominal wall, between the folds of the falciform ligament, which anastomose with the roots of the epigastric and internal mammary veins (*Sappey*).

It is more difficult to explain why some of the symptoms of congestion occur, while others are absent, than it is why none of them exist. We only partly know (see above) why the spleen (which *Bamberger* found enlarged in fifty-eight cases out of sixty-four, and *Frerichs* in eighteen cases out of thirty-six) remains small in some cases, and why some patients have hæmatemesis frequently, while others do not have it throughout the disease; and we shall not attempt to explain these irregularities.

Although, in cirrhosis of the liver, the gall-ducts are subjected to the same pressure as the portal veins, there is rarely much biliary obstruction. It is true, most of the patients have a dirty-yellow color, a yellow tinge of the sclerotic and dark urine; but intense icterus is by no means a frequent symptom of cirrhosis. This symptom is readily explained by the physiology of the formation of bile. There is no bile in the blood going to the liver, but it is prepared there from the materials supplied. Hence obstruction and reabsorption of bile always presuppose that at least part of the liver-cells is preserved, and acts normally. In cirrhosis of the liver, on the one hand, the bile-ducts are compressed, and the conditions are induced which most frequently lead to obstruction and reabsorption of bile; on the other hand, numbers of the liver-cells have been destroyed, and the formation of bile is thus greatly limited. Hence we may readily see why icterus is hardly ever absent in cirrhosis, and, at the same time, why it rarely attains a high grade. Generally, a *slight* degree of icterus, in advanced cirrhosis, is an indication that one factor, the destruction of the liver-cells, prevails; a *higher* grade of icterus indicates that the other factor, compression of the gall-ducts, is in excess, or that, from complication, there is some new obstruction to the flow of bile. These complications, particularly catarrh of the bile-ducts, or their obstruction by gall-stones, occur quite frequently in cirrhosis. If the escape of the bile be entirely prevented, even the slight amount formed by the remaining cells is sufficient to cause intense icterus. The light-gray color of the fæces also depends mostly on the compression of the gall-ducts; as this compression hardly ever causes their absolute closure, perfectly pale, clay-colored stools, such as occur in other forms of icterus, are not seen in cirrhosis. The urine usually contains traces of bile pigment, but is far more remarkable for its richness in urates, and in peculiar coloring matters, to which we shall again refer.

Besides the symptoms due to compression of the portal vein and bile-ducts, there are others which depend on the extensive destruction of the liver-cells. When speaking of the icteroid symptoms, we said that the atrophy of the liver-cells diminished the production of bile; and, probably, the discoloration of the fæces depends as much on limited formation as on retention of the bile. Little as we know of all of the functions of the liver, we are, nevertheless, certain that the formation of bile is not the sole function of the cells of the liver. (The times when fel tauri inspissatum was given in pill, or the patient took fresh ox-gall by the spoonful, "to replace the functions of the liver," are not long past, it is true, but the belief from which such prescriptions started is obsolete.) The liver is very important for the general nutrition, and particularly for the blood, and it is certain that an extensive destruction of liver-cells affects the general health very severely. The affection of the nutrition in patients with cirrhosis of the liver depends partly on the existing gastric and intestinal catarrh; perhaps, also, the excessive fulness of the intestinal veins prevents the entrance of substances from the intestines into these vessels; but there must be another cause for the disturbance of nutrition, for the patients become weaker, more emaciated, and have a dryer skin and more cachectic appearance than those have who are suffering from simple gastric and intestinal catarrh, and in whom the escape of blood from the intestinal veins is obstructed in some other way. Physiology does not, at present, teach us whether the affection of the nutrition depends on arrest of the formation of sugar in the liver, or on the arrest of some other unknown function. In isolated cases, severe brain symptoms appear shortly before death; some patients fall into delirium, and finally into deep sopor; others have symptoms of depression, coma, or sopor, from the commencement. On autopsy, we find no palpable changes in the brain to explain these symptoms; hence we are justified in referring them to an intoxication; but we do not know what substances cause the intoxication. Formerly it was universally supposed that these brain-symptoms depended on the absorption of the constituents of the bile, and they were, consequently, termed *cholæmic* intoxications. But the fact that their frequency is not at all in proportion to the icterus, that, on the contrary, where there is but little jaundice, convulsions, coma, and sopor not unfrequently occur suddenly, while they are often absent in the severest cases where the overloading of the blood with the absorbed constituents of the bile is much more evident, speaks very strongly against the correctness of this explanation. *Frerichs* has advanced the hypothesis that it is not the reabsorption of bile, the so-called cholæmia, which is dangerous, but the acholia, occurring in extensive degeneration of the liver, i. e., that condition where the extensively-diseased liver can no longer prepare bile from the materials supplied to it. When this important process fails, instead of the normal products of interchange of tissue, we have abnormal products of decomposition and poisonous substances. The above severe disturbances of innervation are induced by these substances. The correctness of this hypothesis of *Frerichs* is by no means beyond doubt. When speaking of icterus, we shall return to the relation of the brain-symptoms in question, to cholæmia, or acholia, and show that some recent observers incline to the first theory, as they regard the reabsorbed bile-acids as the poisonous substances.

The occurrence of quantities of abnormal coloring matter, and of urates in the urine of patients suffering from cirrhosis, also appears to depend on the destruction of the liver-cells, and the diminished or altered action of the liver. We do not know what modifications of the change of tissue induce this condition of the urine. The most we can determine is that, if the coloring matter of the urine be derived from the coloring matter of the bile, and this be a derivative from the coloring matter of the blood, in extensive degeneration of the liver, where the coloring matter of the blood is no longer normally transformed into the coloring matter of the bile, this anomaly must influence the formation of the coloring matter of the urine and its modifications.

Lastly, as to the physical signs of interstitial hepatitis, in the first stage, palpation and percussion usually show a very decided increase in size and resistance. In the second stage, also, the liver is not so much out of the reach of palpation as is generally asserted. If, by placing the patient on the left side, and so removing the fluid in the abdomen from the liver, we succeed in reaching the edge of the liver, we perceive that its resistance is even greater than in the first stage, and on the surface we may feel hard, roundish prominences of unequal size. If the ascites be not too great, in the second stage also percussion shows in some cases an increase, in others (but, according to my experience, not at all frequently) a decrease of the normal liver dulness. In estimating the latter symptom, we must be more careful than when the extent of the dulness is abnormally great; for, as the numerous measurements of *Frerichs* prove, the size of the liver and the extent of its dulness vary greatly within certain bounds. Moreover, any abnormal position of the liver, such as occurs by decided inflation of the abdomen, causes the organ to come in contact with the anterior wall of the abdomen and thorax only by its sharp border. Finally, portions of the intestines, filled with gases, pressing between the liver and the abdominal wall, may diminish or entirely remove the normal liver dulness. If we bear these facts in mind, the diminution of liver dulness is a very important symptom in cirrhosis. As the left lobe of the liver is the first to decrease in size, the abnormally clear percussion-sound in the epigastrium is first noticed; subsequently the dulness over the right lobe may so decrease that it will be reduced to one or two inches in the mammillary line (*Bamberger*). The most certain point in diagnosis is the gradual decrease in size of the previously enlarged organ, as shown by repeated examinations.

Having introduced the symptoms of interstitial hepatitis individually, and weighed them as a whole, we will add a short and general description of the disease. The patients are mostly men in middle or advanced life, and addicted to drink. In the commencement the symptoms are slight and obscure; the patients complain of pressure and fulness in the right hypochondrium; more rarely, when the serous covering is more affected and intensely inflamed; there is pain in the region of the liver. In this stage, the most prominent symptoms are enlargement of the liver, dyspepsia, flatulence, and emaciation. Gradually, often not for years, the abdomen swells, from an effusion of fluid into the peritonæum, while there is no simultaneous œdema of the feet. The skin becomes dirty yellow, the urine dark red, and rich in urates, the fæces slate-colored; the dyspepsia and emaciation increase. In this stage, the liver is sometimes smaller, the spleen almost always enlarged. In some patients there is bleeding from the intestinal canal; in almost all, hæmorrhoids. The increasing ascites interferes with breathing, and induces œdema of the legs, genitals, and abdominal walls. Finally, after months or years, the patients die, excessively emaciated and exhausted. During the last days of life, delirium and sopor not unfrequently develop.

Diagnosis.—Cirrhosis would not readily be mistaken for any of the previously described diseases of the liver, but its diagnosis from cancer or tuberculosis of the peritonæum may be very difficult. In these degenerations, as in cirrhosis, there is often ascites, which has been preceded by no other symptoms of dropsy. The patients also soon become emaciated and cachectic, and, as the tumors not unfrequently compress the ductus choledochus, we may have icterus accompanying them. The following points are particularly important in the diagnosis between cirrhosis and these diseases of the peritonæum:

In doubtful cases the dependence of the ascites and other symptoms common to the two diseases on cirrhosis may be suspected—1. When there is swelling of the spleen. We have learned that this is an almost constant symptom of cirrhosis; on the other hand, the spleen is almost always unaffected by tuberculosis and carcinoma, and these are not more likely to cause any other form of enlargement of the spleen. 2. The urine is saturated with abnormal coloring matter, and urates. While this symptom also is rarely absent in cirrhosis, the urine of cancerous or tuberculous patients, like that of all hydræmic persons, is usually very clear and watery. When the degeneration of the peritonæum is accompanied by fever, or when compression of the kidneys and renal blood-vessels by the ascites limits the secretion of urine, the scanty urine may, it is true, be quite concentrated, but there is usually no sediment, and the urine is not so dark as in cirrhosis. 3. The knowledge that the patient was given to drinking. In far the greater number of cases, as we have seen, cirrhosis may be referred to the misuse of spirits, while this has no influence on the development of cancer or tuberculosis.

On the other hand, the following symptoms speak for degeneration of the peritonæum, and against cirrhosis: 1. Extensive sensibility of the abdomen to pressure. 2. Rapid development of ascites. 3. Rapid loss of strength. 4. Recognition of cancer, or tubercles in other organs. 5. Tumors in the abdomen, which may not be felt till after tapping. 6. Occurrence of fibrin, which does not coagulate for a long time, in the fluid evacuated by tapping.

The peculiar color, usual in cancerous persons—which is of some importance in distinguishing cancerous degenerations from other diseases—is of little value in diagnosing carcinomatous degeneration of the peritonæum from cirrhosis; for in the latter also the patient has the dirty-yellow, so-called cancerous hue.

Treatment.—If, as rarely happens, interstitial hepatitis be recognized or suspected in its first stage, we should attempt to arrest its progress by strictly forbidding the use of spirituous liquors. The treatment recommended for hyperæmia of the liver is also suited to these cases, particularly the occasional application of leeches about the anus, and the administration of saline laxatives. The latter are best prescribed as natural or artificial mineral waters of Karlsbad, Marienbad, Tarasp, etc., in which they are better borne than they are without the addition of carbonic acid and the alkaline carbonates. If the nutrition of the patient have already suffered much, we give the preference to springs containing small quantities of iron, such as the Eger, Franzenbrunnen, Kissengen, Ragoczy, and Homburg springs.

In the *second* stage, even at its commencement, we can no longer hope to arrest the disease. As the neoplastic tissue, which fills a loss of substance in the skin, continues to shrink till a firm cicatrix has formed, so the neoplastic connective tissue in the liver unceasingly contracts till the evil results arise which were depicted under the head of symptoms. But then radical aid is entirely impossible, for the dense tissue can never expand again. Subsequently the treatment of cirrhosis can only be symptomatic. Among the symptoms of congestion, the gastric and intestinal catarrh demand particular attention, as they increase the emaciation and debility of the patient. According to the rules previously given, it is just in this form of gastric and intestinal catarrh that the administration of the alkaline carbonates is most beneficial; they appear to decrease the toughness of the mucus, and thus to enable the mucous membrane to get rid of its mucous coating more readily. The hæmorrhage from the stomach and intestines should also be treated according to the rules previously laid down, although we cannot hope for very favorable results. We should only tap the patient when it is imperatively necessary, for the ascites, dependent on obstruction in the portal vein, is particularly liable to return very quickly, as soon as the pressure of the fluid, which has impeded the transudation, has been removed. But, if we have been obliged to tap, we may hope to retard the fresh collection of fluid by compressing the abdomen with a proper bandage. The assertion previously made, that diuretics are as useless as they are irrational in the treatment of ascites, is particularly true of this form of the disease. The most important indication in the treatment of cirrhosis is, to improve the strength and nutrition of the patient. While the state of the digestive organs permits it, we should give him nutritious diet and preparations of iron, which are not unfrequently well borne and very beneficial. In one patient, with cirrhosis of the liver, who afterward died of hæmatemesis, under free use of iron, and a diet consisting mostly of milk and eggs, I have frequently seen the fluid in the abdomen diminish, while it increased again when the patient was removed from the hospital, or was not so well cared for, or when he had hæmorrhage from the stomach.

## CHAPTER IV.

### SYPHILITIC HEPATITIS, SYPHILOMA OF THE LIVER (*Wagner*).

Etiology.—Among the internal organs of the body, the liver appears to be the one most frequently affected by constitutional syphilis. At all events, syphilitic hepatitis or syphiloma of the liver is correctly interpreted earlier than the syphilitic affections of any other organ.

Syphilitic disease of the liver is not unfrequently found in the bodies of children who have had congenital syphilis. Among the disturbances of nutrition due to acquired syphilis, syphilitic hepatitis comes rather late, so that it is classed among the tertiary rather than among the secondary syphilitic diseases.

Anatomical Appearances.—From numerous microscropic examinations of organs affected with syphilitic disease, it is true *Wagner* has come to the conclusion that not only the form appearing as circumscribed deposits (gummy tumors of *Virchow*), but also the diffuse syphilitic degenerations of the organs, depend on the development of a specific neoplasia, syphiloma, but the appearance to the naked eye of livers in which structural change has resulted from constitutional disease varies so greatly, in different cases, that it still appears proper to describe different forms of syphilitic hepatitis. We may distinguish a syphilitic perihepatitis, a simple interstitial syphilitic hepatitis leading to diffuse induration, and a third form called by *Virchow* gummous hepatitis. The latter, whose syphilitic nature was long since recognized by *Dittrich*, is most readily recognized and distinguished from other forms of liver disease. In it we find spots, from the size of a hemp-seed to that of a hazel-nut, or even walnut, in the liver, which in recent cases have a medullary appearance, but, after they have existed a long while, form yellow, cheesy masses. These spots, which, previous to *Dittrich's* explanation, were regarded as cancer in the stage of recovery (which they greatly resemble), are enclosed by a dense tissue, and dense connective-tissue striæ extend from them in various directions toward the surface of the liver. On the surface even we may notice deep furrows, which give the liver a peculiar lobulated appearance, and which are caused by the parenchyma of the liver being destroyed in some places and being replaced by contracting connective tissue. In the diffuse syphilitic indurations of the liver, we find more or less extensive parts transformed into a hard, dense tissue. The gland-substance is mostly destroyed and replaced by connective tissue. The simultaneous occurrence of the above-described spots is almost the only symptom which will prevent our mistaking syphilitic induration of the liver for cirrhosis; but the more regular homogeneous appearance of the cut surface and the absence of the granulations, which are rarely never wanting in cirrhosis, furnish some points for the diagnosis. Besides the fact that *syphilitic perihepatitis* usually complicates the above-described parenchymatous diseases, it is somewhat characteristic of this affection that the thickenings of the serous covering caused by it are more decided than in other forms of perihepatitis, and that they are peculiarly hard and tough.

SYMPTOMS AND COURSE.—In many cases syphilitic hepatitis cannot be recognized or suspected during life. Occasionally we may make the diagnosis from the peculiar form of the enlarged liver, on whose surface prominences and retractions may be distinguished, and from the coexistence of other symptoms of constitutional syphilis. In one patient in Greifswald, who complained of the symptoms of chronic peritonitis, from the peculiar form of the liver I was able to diagnose the probable existence of hepatitis, before the patient acknowledged to being infected, and before examination of the throat had shown a decided defect in both sides of the soft palate. This patient has since died, and, according to a notice that I have found in the *Griefswalder Medicinischen Beiträgen*, the autopsy confirmed my diagnosis. In the former editions of my book I asserted that it was not improbable that, where the process was very extensive, compression of the portal vein and bile-ducts might induce a series of symptoms similar to those from cirrhosis. I was then obliged to add that, in the cases then published, there had been a moderate ascites in only one, while icterus had not occurred in any case. Since then I have had the opportunity of observing one case that has fully sustained my conjecture: A patient, who denied ever having had syphilis, was received into the clinic with icterus, excessive ascites (which required repeated tapping), and very dark urine, which contained quantities of abnormal coloring matter. The liver was enlarged, and on its surface could be felt distinct round protuberances, which were not puffy or in the form of ridges. The diagnosis of carcinoma of the liver, with consecutive closure of the portal vein, was not confirmed by the autopsy. The liver was typically lobulated, its covering much thickened in some places, a large amount of its parenchyma diffusely indurated; deep in the right lobe of the liver were three or four still fresh, medullary-looking gummy tumors.

TREATMENT.—There can hardly be a question of treatment in syphilitic hepatitis, for, even in those cases where the disease is recognized during life, it is only toward its end. We cannot depend on relaxing or removing the shrunken connective tissue by iodine or mercurial preparations, and consequently are limited to a symptomatic treatment.

## CHAPTER V.

### INFLAMMATION OF THE PORTAL VEIN—PYLEPHLEBITIS.

Etiology.—By pylephlebitis we understand not only those conditions where an inflammation of the wall of the vein induces a clot in the portal vein, but also those where coagulation of the contents of the portal vein occurs independently of inflammation in the walls of the vessel.

The first form—primary phlebitis—is far rarer than the latter. Its exciting causes are partly injuries of the portal vein, partly inflammation of the parts about it, which extends to the wall of the vein.

Secondary phlebitis, or, as it is now called, thrombus of the portal vein, cannot always be referred to any evident causes: 1. In some cases it is due to compression of the trunk of the portal vein, by lymphatic glands, caseously or cancerously degenerated, or by thickened or cicatricially contracted peritonæum. 2. In other cases, compression of the branches of the portal vein, as by cirrhosis, so retards the current of the blood, that coagula form in the trunk or ramifications of the portal vein. 3. It appears to result much more frequently from the gradual increase and extension of a thrombus that has formed in some branch of the portal vein. In the same way, where there is thrombus of one of the crural veins, not unfrequently a clot occurs not only in the vein of the corresponding leg, but the thrombus often extends upward also into the vena cava, or even into the renal veins. In such cases there is a primary thrombosis in the portal vein and its branches, even when the original coagulum in one of the roots of the portal vein resulted from inflammation of its walls. In this way are most readily explained the thromboses of the portal vein, due to ulcerations and suppurations in the abdomen, to inflammation of the umbilical vein in newly-born children, to abscesses of the spleen, ulcers of the stomach, to inflamed and suppurating hæmorrhoidal tumors, and to similar causes. 4. It has not been determined whether emboli, from collections of pus reaching the liver, can give rise to a coagulum at first circumscribed, subsequently diffuse, in the portal vein.

Anatomical Appearances.—In the first stage of both forms of pylephlebitis there is always coagulation of the contents of the vein. It is important to note this, to avoid error: suppurative phlebitis begins with suppuration in the vein. The clot adheres firmly to the wall of the vein. In primary phlebitis this is from the first thickened, infiltrated with serum, and shows a cloudiness of the mucous coat, and an injection of the fibrous. In thrombosis, the wall of the vein is at first normal, but it is soon changed in the manner above described. The coagulation of the contents may be limited to some twigs of the portal vein, but in other cases it extends to the trunk, roots, and branches. The terminations of pylephlebitis vary, and, according to the difference of its termination, it is called *adhesive* or *suppurative*.

In adhesive pylephlebitis, while the thrombus gradually shrinks, undergoes fatty degeneration, and is partly or entirely absorbed, there is inflammatory proliferation of the wall of the vein, which terminates in its obliteration, although we cannot follow the different phases of the process. If we examine a liver that has been the seat of adhesive pylephlebitis, we find on its surface cicatricial retractions, and within it, corresponding to these retracted places, we find a hard tissue, in which may still be recognized the atrophied branches of the portal vein. Occasionally these contain remains of the thromboses, colored more or less yellow by hæmatin.

In suppurative pylephlebitis, instead of atrophying gradually, the thrombus dissolves into a puruloid fluid. This is, for the most part, a finely-granular detritus, containing only a few roundish cells, which may be either white blood-corpuscles or newly-formed pus-corpuscles. The whole thrombus rarely breaks down at the same time. In the trunk of the vein there is often a firm coagulum, while there is a puruloid fluid in the branches and roots. But more frequently there is no disintegration in the finer branches of the portal vein, so that the coagula there prevent the disintegrated masses entering the hepatic vein, and reaching the pulmonary circulation. I have had the opportunity of carefully observing this "sequestration" in two cases of suppurative pylephlebitis. It readily explains the frequent escape of the lungs from secondary disease, which could scarcely fail to occur if the terminations of the portal vein were not closed.

But, in phlebitis of the peripheral veins, the inflammation not unfrequently extends from the adventitia to the surrounding parts, inducing suppuration and formation of abscesses, so that inflammation of the parenchyma of the liver, which terminates in the formation of abscesses, often accompanies suppurative pylephlebitis. Then we often find in the liver numerous deposits of pus, which surround the portal vein, and often communicate with it.

SYMPTOMS AND COURSE.—When *adhesive pylephlebitis* is limited to individual twigs of the portal vein, it runs its course without showing any symptoms during life. The pervious branches suffice to transfer the blood from the abdominal organs to the hepatic vein. If the trunk of the portal vein, or all or most of its branches, be obliterated, the symptoms greatly resemble those of cirrhosis. In both cases the obstruction to the escape of blood from the roots of the portal vein leads to gastric and intestinal catarrh and hæmorrhage, to hæmorrhoids, enlargement of the spleen (not constantly), and to ascites. Biliary retention and icterus result more frequently from compression of the gall-ducts in adhesive pylephlebitis than in cirrhosis, because a greater number of the liver-cells are preserved to prepare bile. The continued secretion of bile and the occurrence of icterus in pylephlebitis appear to show that the hepatic artery, as well as the portal vein, furnishes the liver with material for the formation of bile. The course of the disease is chronic. Recovery is impossible; but it often lasts for months before death occurs from the same symptoms as it does in cirrhosis. Hence it appears that the disease can only be recognized and distinguished from cirrhosis by aid of the history of the case. If it be found that the patient was not given to drinking, and if the above symptoms were preceded by chronic inflammation and suppuration in the abdomen, the chances are in favor of adhesive pylephlebitis, particularly if the disease have run its course more rapidly than is customary with cirrhosis.

Hitherto *suppurative pylephlebitis* has rarely been recognized during life. Its symptoms are pain in the right hypochondrium, enlargement and tenderness of the liver, chills recurring at irregular intervals, high fever, and almost always icterus. If these symptoms join themselves to an inflammation or ulceration of one of the abdominal organs, we may, with some certainty, conclude that there is an acute inflammation of the liver; but we cannot yet say whether the parenchyma or the portal vein be inflamed. We are only justified in the latter supposition, when, besides the other symptoms, we have those of obstruction of the portal vein, particularly when there is enlargement of the spleen, slight ascites, and hæmorrhage from the stomach. *Schönlein* was the first to recognize a case of suppurative pylephlebitis during life, from the above symptoms; thereby showing great diagnostic acumen and anatomico-physiological knowledge.

TREATMENT.—Concerning the treatment of adhesive pylephlebitis we may refer to what has been said of cirrhosis; while that of suppurative pylephlebitis corresponds exactly with that of suppurative hepatitis.

## CHAPTER VI.

### FATTY LIVER—HEPAR ADIPOSUM.

ETIOLOGY.—There are two forms of fatty liver. In one, superfluous fat is deposited in the liver-cells from the blood of the portal vein; in the other, the nutrition of the liver-cells is disturbed by disease of the parenchyma of the liver, and they undergo retrogressive metamorphosis, during which fat granules appear in them, as happens under similar circumstances in other cells and other tissues. This second form, *fatty degeneration*, is one symptom of many structural changes of the liver; we have already mentioned it in cirrhosis, and shall often refer to it again. Here we shall only consider the first form, *fatty liver* in the strict sense, or, as we may call it with *Frerichs*, *fatty infiltration*.

On superficial observation, the circumstances under which fatty liver occurs appear very varied: for, on the one hand we find it, along with an excessive production of fat throughout the body, where the supply of nutriment is excessive and its consumption limited; and, on the other hand, it occurs with excessive emaciation, where there is increased consumption of the body. This contrast is, however, only apparent; both circumstances agree in causing an abnormal amount of fat in the liver. In the one case, fat or the substances from which it is formed in the body are supplied from without; in the other case, fat is reabsorbed from the subcutaneous and other tissues rich in fat, and taken into the blood.

If we inquire more minutely into the first-mentioned mode of development of fat, we find that the persons affected with fatty liver are chiefly those who exercise but little, while they eat and drink freely. But by this mode of life they are subjected to conditions analogous to those under which we place animals when we wish to fatten them. We do not let the latter work, but shut them up in a pen, and give them plenty of hydrocarbons. But, under this treatment, one animal will become fat readily and quickly, while another will do so slowly or not at all; in the same way, of two persons living alike, one will become fat and have fatty liver, while the other will remain lean and his liver will be healthy. We do not know the causes of the individual predispositions, which appear to be sometimes congenital and hereditary in some families, or the causes of immunity of other persons to fat bellies and livers. They may depend either on easy or difficult assimilation of nutritive materials; or on slow or rapid consumption of tissue. If there be a decided predisposition, the disease appears to develop on ordinary mixed diet, if more of it be consumed than is required to supply the place of what has been used up; if the predisposition to fat be slight, it only occurs where there is an excessive supply of fats, hydrocarbons, and particularly of spirituous liquors. It is probable, but not absolutely certain, that the latter act by retarding the transformation of tissue.

The frequent occurrence of fatty liver with tuberculosis of the lungs has long been remarked; their connection has been ascribed to incomplete oxidation of the hydrocarbons, and their transformation into fat, due to impaired respiration. But as fatty liver rarely occurs in other lung-diseases where the respiration is also affected, and as it often results from tuberculosis of the bones and intestines, and from carcinomatous and other diseases in which the patients emaciate, the obstructed respiration cannot be the sole cause of its occurrence in tuberculosis of the lungs. *Budd* and *Frerichs* agree with the theory first advanced by *Larrey*, that it depends on too much fat in the blood, and that this was due to the emaciation and reabsorption of fat from other parts of the body. Perhaps the *grade* of the fatty liver is somewhat influenced by the cod-liver oil so much given of late for tuberculosis of the lungs.

ANATOMICAL APPEARANCES.—Slight amounts of fatty infiltration do not alter the size or appearance of the liver, and can only be recognized by the microscope. In higher grades the liver is enlarged, but usually appears flattened; the edges are generally thickened and rounded off. In many cases the increase in size and weight is but slight, in some it is very decided. The peritoneal covering of the fatty liver is transparent, smooth, and shining; occasionally it is traversed by varicose vessels. According to the grade of the fatty infiltration, the surface of the liver is yellowish red, or distinctly yellow. We often notice that the yellow color is interrupted by reddish spots and figures, which correspond to the vicinity of the central veins. The consistence of the liver is diminished; it feels doughy, and pits on pressure with the finger. On incision, we meet little resistance; a coating of fat remains on the warmed knife-blade. But little blood flows from the cut surface, which is also yellowish red or yellow, and shows the red spots and figures above mentioned.

On microscopic examination, according to the grade of the disease, the enlarged and usually rounded liver-cells appear either filled with fine fat globules, or these have united to form single larger drops, or, lastly, individual liver-cells are entirely or mostly filled by one large drop of fat. The infiltration always begins at the periphery of the lobules of the liver, that is, near the interlobular veins, the terminations of the portal vein; it rarely extends to the vicinity of the central vein (whose freedom causes the red spots in the yellow liver), and even then the liver-cells in the centre are usually less infiltrated than those at the periphery. The chemical examination of the liver often shows enormous quantities of fat. In one very fatty liver, *Vauquelin* found 45 per cent. of fat, in one case *Frerichs* found 43 per cent., and when the substance of the liver was freed from water he found 78 per cent. According to *Frerichs*, the fat consists of olein and margarin in variable proportions, with traces of cholesterin.

One variety of fatty liver is what *Home* and *Rokitansky* call *waxy* liver. It depends on the same structural changes, but is distinguished by a waxy dryness, a peculiar brilliance and intense yellow color.

SYMPTOMS AND COURSE.—In most cases of fatty liver there are no subjective symptoms, and, on objective examination also, only high grades of the disease can be recognized. In fat persons, and in those with consumption of the lungs, we should examine the region of the liver from time to time, even without their complaining. If, in these cases, we find an enlargement of the liver, which is the more readily recognized as the liver is usually elongated, has thickened edges, and from the relaxation of its parenchyma hangs far down (*Frerichs*), and if the enlarged liver be painless, its surface smooth, its resistance slight, so that we cannot readily feel the lower border, these symptoms suffice to complete the diagnosis, on account of the frequent coincidence of fatty liver with these states.

If the fatty liver be of high grade, as occurs particularly in topers, as in any other enlargement of the liver, there may be a feeling of fulness in the right hypochondrium. If the abdominal walls, the omentum, and mesentery be also very fatty, the fulness of the abdomen and the tension of its walls may impede the movements of the diaphragm and interfere with respiration. In such persons the secretion from the sebaceous glands is usually so increased that their skin shines with fat, and, when they sweat, the sweat runs from their skin in large pearls; this condition of the skin, which is due to the same state of affairs as the fatty liver, is often mentioned as one of its symptoms.

As fatty livers rarely cause any trouble, as on *post-mortem* examination the bile is usually found in normal amount and quality, as they can be generally well injected, and as there are usually no symptoms of congestion in the abdominal organs, the belief has gradually gained ground that the fatty infiltration neither impairs the functions of the organ nor interferes with its circulation. But this supposition only appears correct for the lower and medium grades of the disease. In the highest grades, after death, we often find but little bile in the bile-ducts, and the fæces in the intestines are but slightly colored. The weakly constitution of such patients, particularly their known intolerance of bleeding, also indicates disturbance of the function of the liver. From the varicosities not unfrequently found on the capsule of the liver, *Frerichs* concluded that the compression of the blood-vessels also caused a slight congestion of the vessels before they enter the liver. It is true there is no enlargement of the spleen or ascites, but the gastric and the intestinal catarrh appear to depend, at least partly, on this congestion. *Rilliet* and *Barthez* consider it not improbable that the profuse diarrhœa which occurs without perceptible structural change of the intestines, in phthisical patients who have fatty liver, is caused by the latter. *Schönlein* and *Frerichs* speak in the same way. I have seen obstinate diarrhœa in non-phthisical patients, where excessively fatty liver was the only anomaly found in the abdominal organs on *post-mortem* examination.

Treatment.—In gluttons and topers, the *causal indications* imperatively demand a change of the mode of life. General advice is of no use, as it is badly followed. For such patients we should prescribe the hours of exercise, forbid afternoon naps, give careful directions about their meals, forbidding all gravies and other fatty substances; for supper we should only allow water-soup and a little stewed fruit. The use of coffee and tea should be limited, that of liquor entirely forbidden. In the fatty liver occurring in consumptive diseases, particularly in pulmonary consumption, we can rarely fulfil the causal indications.

The indications from the disease have long been supposed to require remedies for increasing the secretion of bile. And, in the present state of physiology, we must suppose that the success of this intention would have the best effect on fatty liver. We find less fat in the hepatic vein than in the portal vein. *Frerichs* saw the secretory activity of the liver-cells diminish as their fatty contents increased; hence we can hardly doubt that the fat going to the liver is used up in the production of bile, and that the superfluous fat must disappear from the liver-cells when the secretion of bile is increased. But our knowledge of the difficulty of fulfilling this indication increases in proportion with our comprehension of its urgency. At present we can scarcely hope that an inert indifferent vegetable extract will decidedly increase the secretion of bile, since we no longer regard the bile as a secretion necessary for digestion, or, at least, only secondarily so, but as a product whose quantity and quality vary with the acceleration or retardation of the change of tissue, or with its other modifications. It is possible that the freshly-expressed juices of taraxacum, chelidonium, etc., have a curative influence when used as "spring cures" (*Frühlingscuren*), while the patients rise early, live moderately, and exercise freely; but it is probable that the benefit is mostly due to the change in the mode of life. The case is different with the treatment at Karlsbad, Marienbad, Homburg, Kissengen, etc. In the results there obtained, the favorable mode of life must be taken into consideration; but the free and continued use of the different solutions of salts must have just as much effect on the change of tissue. It is certain that the superfluous fat of the body soon disappears under the use of these mineral waters, and after a month's residence in Karlsbad most patients return home much thinner than when they went there. Simple pedestrian excursions, with the most moderate manner of living, have not, by any means, the same effect. Several very crude hypotheses have been advanced concerning the action of the alkaline-saline springs; the body of a Karlsbad patient has even been compared to a soap-factory, and the characteristic passages have been regarded as soap, which was said to be formed from the soda of the waters and the fat from the body. We should not, however, wait to find a better explanation, but should send fatty patients with fatty infiltrations of the liver to those watering-places. This is occasionally very erroneously done with patients whose fatty liver depends on decided emaciation, because the patients or the physician do not recognize the significance of the disease. It is unnecessary to state the contraindications to the use of the alkaline-muriatic springs. If the blood be impoverished, we should carefully try if the Eger Franzensbrunnen, or the Kissengen Ragoczy waters, are borne, and, if they are not, we should be satisfied with regulating the diet and mode of life. This rule refers also to those cases where patients with fatty liver are inclined to diarrhœa.

## CHAPTER VII.

### LARDACEOUS (OR WAXY) LIVER—AMYLOID DEGENERATION OF THE LIVER—(*Virchow*).

Etiology.—Lardaceous degeneration of the liver depends on a deposit in the liver-cells and in the walls of the hepatic vessels (*Wagner*) of a substance whose nature we do not yet know, but whose reaction to iodine and sulphuric acid closely resembles that of amylum and cellulose. From this similarity of chemical reaction, which may, perhaps, be accidental, the title of "amyloid degeneration" has of late been given to that state which was formerly called "lardaceous degeneration," from its external resemblance, but particularly from its peculiar lustre.

Lardaceous liver never occurs in persons otherwise healthy; it is more apt to occur in advanced cachexia, particularly in cases resulting from scrofulous, cachectic, or syphilitic affections, from mercurialism, tedious suppurations, and caries of the bones; it is also occasionally found in patients with pulmonary consumption; in some cases it is induced by malaria.

Anatomical Appearances.—A lardaceous liver is usually decidedly increased in size and weight, and resembles fatty liver in form, as it appears elongated, flattened, and thickened at the edges. The peritoneal covering is smooth and tense, and the liver is hard as a board. The cut surface is very dry and bloodless, smooth, almost homogeneous, with a gray color and very lardaceous lustre. When there is a coincident fatty degeneration, the knife-blade is covered with fat. There is almost always a similar degeneration of the spleen, and not unfrequently of the kidneys also.

On microscopic examination, the polygonal liver-cells appear round and enlarged; the fine granular contents, and usually also their nuclei, are atrophied, and the cells filled with a translucent, homogeneous substance. If there be at the same time fatty degeneration, we find small discrete fat globules in the degenerated cells, particularly at the periphery of the lobules of the liver. On the addition of a solution of iodine, there is not a yellowish-brown but a peculiar reddish-brown color; after the addition of sulphuric acid, there is a violet and subsequently a blue color of the preparation.

SYMPTOMS AND COURSE.—The very gradual enlargement of the liver causes no pain; and the patient's attention is first called to his disease, when the enlarged organ fills the right hypochondrium, and causes a feeling of pressure and tension. *Budd* considers ascites as a constant symptom of lardaceous liver, and refers it to the compression of the portal vessels. He also believes that, in children debilitated by scrofulous diseases of the glands and joints, the recognition of a painless enlargement of the liver, accompanied by ascites, is sufficient for the diagnosis of the disease in question. In opposition to the view that ascites accompanying lardaceous liver is due to obstruction of the portal vein, *Bamberger* very correctly says that in such a case there should also be symptoms of congestion in the other abdominal organs, but these never occur. It is far more probable that the dropsy is due to the general cachexia and hydræmia, from which all patients with lardaceous liver suffer. In the cases observed by *Bamberger*, the ascites was always preceded by œdema of the feet, and in those related by *Budd* it does not appear that the ascites preceded the œdema of the feet. The enlarged liver-cells do not compress the bile-ducts any more than they do the blood-vessels, and icterus is absent as a rule. Icterus may, however, result from complications, such as lardaceous enlargement of the lymphatic glands at the porta hepatis, so that *Frerichs* warns us against considering the absence of icterus as a diagnostic criterion of lardaceous liver. The fæces have little color, on account of the impaired function of the diseased liver-cells. It is difficult to decide how far the bad nutrition of the patient, the paleness of his skin and mucous membranes, the hydræmia and dropsy, depend on the degeneration of the liver, as this disease only occurs in those who are cachectic at any rate, and as the spleen is almost always diseased at the same time, and the kidneys are very frequently so. The etiology, the hard liver-tumor readily felt on palpation, the usually coincident enlargement of the spleen, and, lastly, albuminuria, when it exists, are important in the diagnosis of lardaceous liver. By paying attention to these points, the higher grades of the disease may be readily recognized.

TREATMENT.—It has not been proved, nor is it probable, that lardaceous degeneration is capable of restoration; and, although cases are said to have been observed where lardaceous livers have become smaller and normal, further proof is needed on this subject before we can believe the statement. The long-continued inunction of iodine salve over the liver, although strongly recommended by *Budd*, deserves little confidence. The preparations of iodine, particularly syrupus ferri iodidi, are extensively used in lardaceous liver, as are also alkaline baths and preparations of iron. Although these may not improve the liver-disease, they may do much to arrest its progress. Iodine justly holds the reputation of being a specific for tertiary syphilitic affections, and in other dyscrasias also its beneficial effects have been proved; the preparations of iron are indicated by the great poverty of the blood. The peculiarities of each case should decide which of these remedies is to be employed.

## CHAPTER VIII.

### CANCER OF THE LIVER—CARCINOMA HEPATIS.

ETIOLOGY.—The liver is so frequently affected with carcinoma that, according to *Rokitansky*, there is about one case of cancer of the liver to every five cases in all parts of the body, and *Oppolzer* found it fifty-three times in four thousand autopsies, or in about every eightieth patient. In many cases it is primary, in others it is preceded by cancer of the stomach, rectum, or other organs; it is peculiarly apt to develop after extirpation of peripheral cancerous tumors.

The causes of carcinoma of the liver are just as obscure as those of carcinoma elsewhere. It is true that, when asked, the patients are rarely puzzled to tell what caused their disease, but their accounts give us no true information as to its etiology.

ANATOMICAL APPEARANCES.—*Medullary* cancer is the form most frequently found in the liver. It sometimes forms circumscribed, sharply-bounded tumors; sometimes it spreads out diffusely between the liver-cells, and has no sharp borders.

In the former case we find roundish or glandular and lobulated tumors in the liver; these are enclosed by a delicate, vascular connective tissue capsule, and, where they touch the peritonæum, are often flattened, or have a shallow excavation, a so-called "cancer navel." The size and number of the tumors vary; they are found from the size of a pea to that of a child's head; sometimes they are solitary, again innumerable. The nearer they lie to the periphery of the liver, the more readily do knobbed protuberances appear. Their consistence varies from that of firm brawn to that of soft brain-matter. A large amount of "cancer-juice" may be pressed out of the softer cancers, while only a small amount can be expressed from the harder ones. Lastly, the color of the tumor is milk-white or reddish, according as it has few or many vessels; it may also be dark red, from effusions of blood, or black from deposits of pigment. In the unaffected parts of the liver there is usually great hyperæmia, which has something to do with the enlargement of the organ, which is often very great. Not unfrequently the liver is rendered intensely yellow by compression of the gall-ducts and retention of the bile. In the immediate vicinity of the cancerous tumors, the liver-cells have usually undergone fatty degeneration. Chronic partial peritonitis almost always occurs quite early in the covering just over the tumors, causing thickening and adhesion with the neighboring parts; in other cases cancerous masses develop in this part, and spread over the entire peritonæum. The formation of the "cancer navel" in carcinoma of the liver, as in carcinoma elsewhere, depends on atrophy of the oldest parts of the neoplasia, in which the cellular elements undergo fatty degeneration and atrophy; but occasionally we meet cases of cancer of the liver where this retrogression extends to the entire tumor, which is finally reduced to a yellow crumbly mass, enclosed in a cicatricial connective tissue (the remains of the cancerous framework). If young cancer be found with these cicatricial masses in the liver, there can be no doubt about the nature of the latter; but if this be not the case, it will be difficult to decide whether it be indeed cancer that has recovered or the remains of some other process. In rare cases medullary cancer softens, and, by disintegrating, leads to acute peritonitis, or to dangerous hæmorrhage.

In the second form, which *Rokitansky* calls *infiltrated cancer*, we find large portions of the liver converted into a white cancerous mass. The obliterated vessels and gall-ducts, which are surrounded by rudimentary liver-cells atrophied, fattily degenerated and colored by bile, often traverse this white mass as a coarse yellow framework. Toward the periphery the infiltrated cancer gradually passes into the normal parenchyma, as there are places where cancerous masses, and others where the liver-cells, are in excess.

The *alveolar* or *gelatinous* cancer, which almost exclusively attacks the stomach, intestines, and peritonæum, in some few cases extends from the latter to the parenchyma of the liver. In one case, observed by *Luschka*, almost the whole of the liver was transformed into a shapeless mass of the structure of alveolar cancer.

Still more rarely small nodules of the structure of epithelial cancer are found in the liver.

Occasionally cancer of the liver is accompanied by cancer of the portal vein, the trunk, roots, and branches of the latter being filled by a loosely-connected thrombus of cancerous substance. I have seen one case where the contrary occurred: cancer of the portal vein first complicated cancer of the stomach, and the cancer of the liver found on autopsy had evidently resulted from the extension of the degeneration from the portal vein to the tissue of the liver.

Symptoms and Course.—The symptoms of cancer of the liver are always obscure at first; later they are usually quite distinctive, but cases do occur where a certain diagnosis cannot be made till death. The first complaints of the patient are almost always of a feeling of pressure and fulness in the right hypochondrium, such as accompanies all enlargements of the liver when they occur rapidly and become great. When the tumors are near the surface of the liver, and hence induce partial peritonitis early, there is pain in the region of the liver even at the commencement of the disease; this pain often spreads to the right shoulder. Even from the first the region of the liver is usually more sensitive to pressure than in any of the diseases of the organ hitherto described, except suppurative hepatitis. After a time the patients themselves notice that their right side is prominent, and that there is a hard tumor in the right hypochondrium. If the tumors compress large branches of the portal vein, there is moderate ascites; if, on the other hand, they grow on the concavity of the liver and compress the portal vein itself, the ascites becomes considerable; in other cases there is none, but this is rare; for, besides the obstruction of the vessels, consecutive disease of the peritonæum causes ascites. Gastric and intestinal catarrh, which often complicate cancer of the liver without the stomach or intestines being affected with cancer, are to be regarded as due to the obstruction of the circulation. The spleen is rarely enlarged, perhaps because the hydræmia favors the early occurrence of dropsy, and the pressure of the dropsical fluid interferes with the swelling of the spleen. The same is true of icterus as of ascites. Compression of the large bile-ducts causes partial obstruction of bile and moderate jaundice; but, from the gall-ducts that are not compressed, sufficient bile flows into the duodenum to color the fæces normally. If the ductus choledochus be compressed, however, the obstruction of bile becomes general, the icterus great, and the fæces are colorless. Lastly, the jaundice and discoloration of the fæces occasionally depend on catarrh of the gall-ducts. In more than half of the cases there is no jaundice. As icterus does not occur in most of the organic diseases of the liver, its presence with enlargement of the liver is strong evidence that this is due to carcinoma rather than to other diseases; but its absence cannot be regarded as proof against carcinoma. Where the cancer of the liver is extensive, the urine shows the peculiarities before mentioned, and appears peculiarly red or bluish from the presence of abnormal coloring matter, resulting from the destruction of numerous liver-cells.

While the above symptoms gradually occur, the diseases may generally be suspected from the appearance of the patient, who looks cachectic, is wasted away, has a relaxed skin, and œdema about the ankles. In some patients the cancerous marasmus does not appear till very late, and, even when large tumors may be felt in the liver, they appear as well nourished and fresh as many patients with carcinoma of the breast, before it has ulcerated. But even these patients do not escape the injurious effects on the general health, which are difficult to understand when the cancer does not suppurate. They gradually become marasmic; and, when the emaciation and exhaustion have reached the highest grade, they generally die of dropsy. Among the final symptoms we not only unfrequently have thrombus of the femoral veins, follicular catarrh of the large intestine, and shortly before death thrush not unfrequently comes in the mouth.

Where there are large tumors in the liver, the physical examination not unfrequently gives important aid in diagnosis. In none of the diseases of the liver, so far described, does the organ attain the size that it may reach in carcinomatous degeneration. The liver, enlarged by this disease, most frequently elevates the lower ribs, pushes them outward, and forms a visible prominence in the abdomen, which often has the outline of the liver, and may extend from the right hypochondrium to below the navel and into the left hypochondrium. On palpation, we usually distinctly feel the edge of the indurated organ, and on its surface we find larger or smaller protuberances, which are almost pathognomonic of the disease.

If the peritoneal covering of the tumor be the seat of a recent inflammation, we sometimes feel and hear a distinct friction from the movements of the liver during respiration. If there be considerable ascites, it may prevent exact examination of the surface of the liver; but if, by pushing the finger in quickly, we displace the fluid, we may at least convince ourselves of its increased size and consistence.

In most cases the above-described symptoms and course render the diagnosis of cancer of the liver quite easy; but, as we have before said, it is occasionally difficult or even impossible. Where the cancer is infiltrated, or a few small cancerous nodules develop deep in the liver, this is often only slightly enlarged, and even when it projects from beneath the ribs we do not find the characteristic peculiarities of its surface, on palpation. There is little or no pain, as the serous covering is not inflamed, and there is usually no ascites or icterus, as neither the branches of the portal vein nor the gall-ducts are much compressed. In such cases we often have no sufficient grounds for suspecting the disease, till our suspicion of cancer is awakened by a gradually increasing cachexia, for which we can find no other explanation, as all the functions are undisturbed; and when cancer of the uterus, stomach, etc., where it is more readily recognized, can be excluded. The probability is still greater when the suspicious marasmus has developed after the operative removal of an external cancer. If, besides the cancer of the liver, which runs its course without decided enlargement and pain of the liver, or icterus, or ascites, there be cancer of the stomach, morbus Brightii, or some other disease, to explain the marasmus, the disease may often escape suspicion.

TREATMENT.—We cannot expect any successful radical treatment of cancer of the liver. In most cases we must satisfy ourselves with maintaining the strength of the patient as long as possible by careful nourishment. If the liver become very painful from intense hepatitis, we apply a few leeches, and cover the region of the liver with warm poultices; these means almost always remove or at least relieve the pains. The ascites complicating cancer of the liver may require tapping under the previously-mentioned conditions.

## CHAPTER IX.

### TUBERCULOSIS OF THE LIVER.

TUBERCULOSIS of the liver is never primary, but always accompanies an already existing tuberculosis of other organs, or else forms one symptom of miliary tuberculosis. In the latter case, we only find dull, translucent granulations, as large as grains of sand, which occur particularly on the surface of the liver, with advanced tuberculosis of the intestines and lungs. On the other hand, we occasionally find yellow, cheesy, tuberculous masses, as large as a hemp-seed, pea, or even larger. These very rarely break down into small vomicæ, filled with tuberculous pus. But they often compress capillary bile-ducts, and lead to their dilatation behind the compressed parts; thus forming cavities as large as a hemp-seed or pea, which are filled with bile and mucus, and which we must not confound with tuberculous cavities. Tuberculosis of the liver cannot be recognized during life.

## CHAPTER X.

### ECHINOCOCCI OF THE LIVER.

Etiology.—Echinococci hold the same relation to tænia echinococcus (*Siebold*) that cysticercus cellulosæ does to tænia solium; i. e., they are the young, sexless brood of the mature tape-worm. Experiments of feeding animals with echinococci from man have given no decided results, it is true; but the tænia echinococcus has been found in the intestines of animals that had been fed on echinococci from other animals.

It is doubtful how the egg and embryo of the tænia echinococci reaches the human liver, there to develop to echinococcus vesicles. In Iceland they are so common, that physicians there say that one-eighth of all the diseases of that island are due to this disease, and that about every seventh person contains echinococci (*Küchenmeister*). From analogy, it is supposed that the migration takes place as follows: Animals affected with the tænia echinococcus evacuate mature links from the bowels; the eggs or embryos contained in these in some way get into the drinking-water, or come in contact with some food that is eaten raw. Entering the intestinal canal with these, the small embryos with their six hooks bore into the wall of the stomach or intestine, and, wandering farther, they finally reach the liver. There the microscopic embryo swells to a large vesicle, on whose inner wall a colony of young, immature tæniæ or scoleces is developed. In most cases, besides the scoleces, daughter vesicles develop in the mother vesicle, or rather wet nurse; in these, a second generation of vesicles is formed, whose inner wall is also covered with scoleces.

*Küchenmeister* refers the endemic occurrence of echinococci in Iceland principally to the number of dogs kept there, and to the warmth of the river-water, which is much used for drinking. The dogs probably eat the vesicles that have been evacuated from the mouth, anus, or suppurating sacs that have not been taken care of. The warm temperature of the water is favorable for the embryos of the echinococcus, as it is for all the lower animals. *Küchenmeister* considers it as not improbable that, when the echinococci reach the bowels of the person in whom they exist, they there develop to tænia, and conversely that the embryo escaping into the intestines of persons affected with tænia may become echinococci.

Anatomical Appearances.—Echinococcus sacs are sometimes solitary, sometimes very numerous in the liver, and occur more frequently in the right lobe than in the left. Their size varies from that of a pea to that of a fist or a child's head. If they are large and numerous, the liver is usually decidedly enlarged. Sacs, deep in the organ, and surrounded by liver parenchyma, change the fôrm but little; very large sacs, or those near the surface, are generally elevated above the liver, and cause decided deformity of the organ. Over the peripheral cysts the peritoneal covering of the liver is decidedly thickened, and is attached to the parts around by firm pseudo-membranes. The parenchyma of the liver is displaced by the parasites, and, when these are large and numerous, it is extensively destroyed; the parenchyma still preserved not unfrequently appears very vascular, as a result of partial congestions. The echinococcus vesicle itself is enclosed by a hard fibrous capsule, which is formed by proliferation of connective tissue, but may readily be removed from this. The envelope of the vesicle itself is a delicate, half-transparent membrane, resembling coagulated albumen, which, by the microscope, is shown to consist of numerous fine, concentric lamellæ. If we open the vesicle, a clear serous fluid escapes; this almost always contains numbers of smaller vesicles. The fluid contains about 15 parts of firm constituent to the 1,000; no albumen, but chiefly salts, mostly chloride of sodium, and, according to *Heintz*, 3 parts of succinate of soda to the 1,000. The daughter vesicles are like the mother sac; they are as large as a hemp-seed or a large hazel-nut. The larger ones float about freely in the mother sac; the smaller ones are firmly attached to its inner wall. The grandchild vesicles, which are only found in the larger daughter vesicles, are usually about the size of a pin's head. On careful examination, we may discover a whitish, gritty coating on the inner surface of the mother, daughter, and grandchild vesicles. The microscope shows this to be a colony of young, immature tæniæ or scoleces. The individual animals are about ¼ mm. long, and ⅛ mm. broad; they have a thick head, with four suckers, and a snout which is surrounded by a double row of hooks. The head is separated by a constriction from the short body, in which there are numerous round and oval chalk concrements. The head is generally drawn into the body. The animals are then usually round or heart-shaped, and the circle of hooks is in the middle. At the posterior end of the body is a short pedicle, by which the animal is firmly held till he subsequently breaks loose and floats about in the fluid. The echinococci often die. The mother and daughter vesicles collapse, their contents become cloudy, fatty, and are finally transformed into a smeary or putty-like substance. This consists of chalky salts, fat, and cholesterin, and only a few of the hooklets of the echinococcus remain to betray the origin of the mass. *Budd* compares these hooks to the bones and teeth remaining after the decay of larger animals.

In other cases the echinococcus sac gradually distends till it finally bursts. If the distended and thinned peritoneal covering ruptures at the same time, the contents of the sac enter the peritoneal cavity, and there is severe peritonitis. In the same way, if the sac has become adherent to the neighboring parts, it may be evacuated into the stomach, intestines, gall-ducts, neighboring blood-vessels; or, when the diaphragm has been gradually thinned, and finally perforated by the pressure, the sac may empty into the pleural cavity or into the lung adherent to the pleura. In still other cases, the hydatid excites intense inflammation in its vicinity, particularly in the fibrous envelope belonging to the liver. This appears to occur particularly in those cases where the sac bursts inside of the liver, and its contents come in direct contact with its parenchyma. In such cases, besides shreds of the mother vesicle, and sometimes single, still perfect daughter vesicles, the cyst contains purulent masses tinged with bile. These cases are evidently not due to inflammation of the mother vesicle, but the pus has entered the sac from without. The abscesses of the liver, thus induced, may have any of the terminations described in Chapter II. If it perforates externally, rudiments of the echinococcus vesicle are mingled with the pus that escapes.

Symptoms and Course.—As a rule, echinococci inhabit the liver for years before they attract attention, or the disease is suspected. The gradual growth sufficiently explains the absence of inconvenience, or its tardy appearance. In most cases where the disease is recognized, its discovery is not brought about by the subjective symptoms, but by the patient himself, or the physician accidentally noticing that the right hypochondrium is prominent, and contains a tumor. If the hydatid, and with it the liver itself, attain a considerable size, the feelings of pressure and tension in the right side, so often mentioned, are occasionally induced. The diaphragm, pressed upward, may have its action interfered with. The compression of the lower lobe of the right lung and the collateral hyperæmia in the non-compressed portions of lung may induce dyspnœa and bronchial catarrh. In the same way, ascites and icterus of variable amount may result from compression of the branches or trunk of the portal vein of the small ducts, or the excretory duct of the bile; but all these symptoms are exceptional.

Physical examination is the most important, and in the majority of cases is the sole means of diagnosis. Like large and numerous carcinomatous tumors, large and numerous hydatids in the liver are also often evident on inspection. In these cases also there is a prominence in the right hypochondrium, extending below the navel, and into the left hypochondrium; and, while the shape of the swelling reminds us of the liver, we notice on it slight elevations of different sizes. At the same time, the right half of the thorax may be dilated, the lower ribs being moved upward and outward. On palpation we may still more distinctly recognize the enlargement of the liver, and the inequality of its surface. The protuberances appear more yielding than those due to the softest forms of cancer. Occasionally there is distinct fluctuation. The percussion-sound is absolutely dull all over the enlarged liver. On percussing over the hydatid itself, in some cases we notice a peculiar thrill (*Piorry's* Frémissement hydatique), similar to that obtained by striking on tolerably stiff glue.

Among the symptoms of the termination of the disease, those of the gradual atrophy of the sac cannot be given, as this termination only occurs in small hydatids which cannot be diagnosed. If the hydatid burst into the peritoneal sac, we have the same symptoms as in perforation of ulcers of the the stomach. If the hydatids were not previously diagnosed, we cannot tell what substance has entered the peritonæum. The patients die in a few days of the rapidly-fatal peritonitis. We can only recognize perforation into the stomach, intestines, or lung, when portions of the hydatid are vomited, evacuated at stool, or coughed up. If the echinococcus vesicle excite inflammation in its vicinity, the enlargement of the liver, previously free from pain, now becomes very painful, and is especially very sensitive to pressure. There are chills and high fever, and we have the picture of suppurative hepatitis, and its results, which were described in Chapter II. If the abscess of the liver perforate externally, we may occasionally find traces of the peculiar laminated membrane, or some of the hooks of the hydatid in the pus.

TREATMENT.—Fomentations of strong solutions of common salt over the region of the liver have been recommended for hydatids of that organ, and *Budd* says that, from the peculiar attraction and affinity of the hydatid cyst for salt, it is possible that the collection of the latter in the fluid thus effected may prevent the further development of the echinococcus, or destroy it altogether. Others recommend preparations of iodine and mercurials, on account of their known "anti-parasitic" effect, and anthelmintics are used for the same reason. These remedies deserve little confidence, as they have been advised on theoretical grounds, and not from actual experience. If we conclude to use them, we should at least choose those least injurious to the organism. In Iceland they appear to open hydatids boldly; in this country very bad results have occurred from opening them without precaution; and, when we decide on puncturing them, the same rules are necessary as in opening abscesses of the liver.

## CHAPTER XI.

### MULTILOCULAR HYDATIDS.

ETIOLOGY.—Recently, on *post-mortem* examination, large portions of the liver have been found transformed into a peculiar mass, which consisted of a connective-tissue stroma, and numerous large and small cells filled with a gelatinous substance. The first observers considered these growths as alveolar carcinoma; but careful microscopical examination showed that the gelatinous contents of the cells consisted of the tissue peculiar to the echinococcus, and hence placed it beyond doubt that the affection was due to a brood of hydatids.

It is difficult to determine the mode of origin of these tumors, which, according to *Virchow* are designated multilocular echinococcous tumors; but it is most probable that this is not a peculiar species of parasite, but only that there is a peculiarity about the migration, seat, and growth. *Virchow* believes that this form of the disease results from the echinococcus embryo entering the lymphatics of the liver, and their sacs developing there: *Leukart* locates these processes in the blood-vessels; *Friedreich*, who, in one case found the hepatic duct plugged with echinococci, locates them in the gall-ducts. I am indebted to a letter from *Küchenmeister* for the following simple explanation, which I believe to be the correct one: "After the emigration of an echinococcus embryo, instead of the usual form of the disease, we have a multilocular hydatid cyst, if no connective-tissue sac form around the embryo, or if this sac be ruptured by the parasite before it has become hard and resisting. When the echinococcus has no firm envelope, it can grow freely in all directions, and spreads particularly in the directions where it finds least resistance. If in its wanderings it has entered one of the many channels that traverse the liver, or if it has subsequently broken through the wall of one of these canals, it advances along it, and may finally fill the entire canal system thus affected. As *Virchow*, *Leukart*, and *Friedreich*, who are certainly trustworthy observers, reached different results, as each of these observers found different canal systems of the liver filled with echinococci, we are justified in supposing that the emigrations and perforation of the parasites may occur into the lymphatic, or blood-vessels, or into the bile-ducts, but that each of these canal systems may remain free from them.

ANATOMICAL APPEARANCES.—Multilocular hydatids almost always occur in the right lobe of the liver; in only one of the three cases that I have seen was the left lobe affected. They may reach the size of a child's head or become even larger. The connective-tissue stroma is generally far advanced in fatty metamorphosis. The cells opened by an incision strongly remind us of the holes on the cut surface of a piece of well-baked black bread. On microscopic examination of the gelatinous substance contained in them, we immediately recognize the characteristic membrane of the echinococcus, which is strewn with numerous chalk concrements. On the other hand, it usually requires a long search to discover a circlet of hooks or single members from it, and we rarely find perfect scoleces. In a single one of my cases, at the periphery of the tumor there were vesicles as large as a cherry, whose inner wall was thickly covered with a colony of well-preserved scoleces. In all reported cases, except my last one, the centre of the mass had suppurated; the cavity resulting from the suppuration contained a dirty brownish-gray fluid, which consisted mostly of detritus masses, chalky concrements, fat globules, and cholesterin crystals. The wall, rendered uneven by numerous small fossæ (opened alveoli), had in many places an ochre-colored coating in which beautiful hæmatoidin crystals could be seen microscopically.

SYMPTOMS AND COURSE.—Of course the symptoms of multilocular hydatids must vary with the canal system of the liver, which is filled and obstructed with the echinococcous masses. This explains the remarkable fact that the somewhat hasty description of the symptomatology given by *Friedreich* of the multilocular hydatid cyst so exactly suits some cases, that the disease has been repeatedly diagnosticated from that description; while in other cases even the most prominent points of his description have been wanting. I shall try to avoid *Friedreich's* errors in the following description, which is taken partly from my own comparatively numerous observations, partly from a careful analysis of the reported cases of other observers, which are not very numerous:

The disease is almost always latent at the commencement; as a rule, the first symptoms appear after it has made considerable progress. Some patients have their attention called to the disease by a feeling of pressure and fulness in the right hypochondrium, or by the accidental discovery that they have a tumor in the abdomen. They have nothing else to complain of; the appetite and digestion are good; the strength and nutritive condition leave nothing to desire; there is no jaundice or symptoms of obstruction in the roots of the portal vein. On examining the abdomen, we find in the right hypochondrium a tumor, which unmistakably belongs to the liver; the liver may either retain its normal shape, or there may be slight elevations on its surface, such as occur in carcinomatous and syphilitic diseases. Even where there is extensive central suppuration, the resistance of the liver tumor is usually very decided; fluctuation was only noticed in one of the cases observed by *Griesinger.*

When the disease commences and runs its course with the above symptoms, it can never be recognized with certainty, or absolutely distinguished from other diseases of the liver, particularly carcinomatous or syphilitic. In the first case I saw, death was caused by apoplexy; the preparation was presented to me as an immense suppurating cancer of the liver. The attending physician had not made out the nature of the nodular, stony tumor of the liver, whose gradual growth he had watched for years. The patient had no icterus during the latter years of his life; he only had a slight jaundice, of short duration, about ten years before his death. In a second case, carefully observed for several months, at my clinic, besides the above symptoms, there were albuminuria and general dropsy; the case was diagnosed as syphiloma of the liver and amyloid degeneration of the kidneys. Only the second half of the diagnosis was confirmed by the autopsy. Instead of a syphiloma, the liver contained a multilocular hydatid tumor, as large as the head. This patient was never jaundiced. In both patients the passages and excretory bile-ducts were entirely pervious.

These observations not only disprove *Friedreich's* assertion, that marked icterus is among the most constant symptoms of multilocular hydatid, but they also prove that, in cases where there is no obstruction of the bile-ducts by echinococci, and, consequently, where there is no obstruction and reabsorption of bile, the patient's state may long remain as endurable as in the ordinary form of hydatid. After a long time, the advancing suppuration of the tumor and the fever accompanying it appear to impair the nutrition, and to develop a cachexia, which finally carries off the patient, if he does not die of some intercurrent disease. In my second case, death was hastened by the secondary disease of the kidneys (whose occurrence, in multilocular hydatids, *Friedreich* expressly denies).

The symptoms and course of the disease are entirely different when the bile-ducts are obstructed by echinococci locating in them, or breaking into them from without. In these cases, at least in their later stages, the disease is not unfrequently so characteristic that an approximate, or even an absolute, diagnosis may be made. The series of symptoms is opened by an apparently inoffensive icterus, but this steadily increases; all remedies used for it prove unavailing, and it gradually becomes excessive. The fæces usually soon lose their color, a proof that all the gall-ducts or the excretory ducts are closed. As there are no dyspeptic symptoms, and as the occurrence of the icterus is preceded by no severe paroxysms of pain, we may, with great probability, exclude catarrh of the excretory bile-ducts and their obstruction by calculi; but the true ground of the obstruction is at first entirely obscure. In one patient, where there was finally no doubt about the diagnosis, at the first consultation, I was obliged to limit myself to saying, "There is an obstruction of the excretory bile-ducts, from some cause unknown to me." Early in the disease, on examining the abdomen, we find the liver enlarged; but even this symptom does not enable us to make a diagnosis until the enlargement has become very marked, or the resistance of the liver has greatly increased, and while its surface remains smooth. Where the fæces are discolored and the icterus excessive, we are most apt to ascribe the enlargement of the liver also to biliary obstruction. On the other hand, if, while there is excessive and obstinate icterus, and complete discoloration of the fæces, we find enlargement of the liver, but not of the gall-bladder, we should suspect multilocular hydatids. Under such circumstances, the obstruction is, most probably, not in the ductus choledochus, but in the ductus hepaticus, for when the former is obstructed, the gall-bladder also is usually distended by the obstructed bile. Obstruction of the hepatic duct is rare, but probably its most frequent cause is multilocular hydatids, and hence, when we think we have found it obstructed, we should suspect this disease. The suspicion that there is a multilocular hydatid cyst in the liver increases, and may become a certainty, if, besides the above symptoms, the liver becomes uneven and nodular as the disease progresses. From that time swelling of the liver, from simple biliary obstruction, may be excluded; the case can only belong to one of the varieties of liver-disease where the form of the organ is thus changed—that is, it must be cirrhosis, syphilitic, or carcinomatous disease, or hydatids. The rest of these diseases are hardly ever accompanied by complete discoloration of the fæces, and excessive and obstinate biliary obstruction, in which the gall-bladder does not participate, while these are very frequent accompaniments of multilocular hydatids. Hence, if we find a hard nodular enlargement of the liver *with* those symptoms, we are justified in diagnosing a multilocular hydatid.

In many cases, in the course of the disease, there are ascites, enlargement of the spleen, gastric and intestinal hæmorrhage, occasionally also hæmorrhage from other mucous membranes, and effusion of blood into the cutaneous tissue. But none of these symptoms are pathognomonic of multilocular hydatid; they result from the great biliary obstruction and reabsorption of bile, and, as we shall show in the next chapter, occur just as often in other forms of obstinate obstruction and reabsorption of bile.

TREATMENT.—Treatment can accomplish nothing in multilocular hydatids; occasionally the inefficiency of the remedies used for the icterus strengthens the diagnosis. Of course, there is no way of reducing the size of the tumor, or of improving the jaundice and inconveniences dependent on it, or of permanently maintaining the strength and nutrition of the patient. Nor does the attempt at tapping, made in *Griesinger's* case, encourage imitation.

## CHAPTER XII.

### BILIARY OBSTRUCTION IN THE LIVER, AND CONSEQUENT ICTERUS—HEPATOGENOUS ICTERUS [JAUNDICE].

ETIOLOGY.—The gall-ducts have no contractile elements to urge their contents onward. Hence we are led to the conclusion that the bile in the biliary passages is pressed forward by the same force that caused it to enter the ducts, the secretory pressure. The compression to which the liver is subjected, from the descent of the diaphragm during inspiration, assists the evacuation of the bile-ducts, it is true, but we should not over-value this force, for the gall-bladder, on which the pressure must be greater than on the firm liver, may be greatly distended with bile, while the movements of respiration go on uninterruptedly. At all events, the forces that pass the bile along its ducts are so weak that they cannot readily overcome even the slightest obstacle, and a very inconsiderable obstruction to the evacuation of the bile suffices to cause it to collect in the liver—that is, to induce retention of bile.

If the bile-ducts and the liver-cells become very full, and the lateral pressure in them attains a certain height, a large part of their contents enters (filters into) the blood-vessels and lymphatics. This is the most frequent cause of jaundice. Recent investigations have placed it beyond a doubt that, in the icterus due to obstruction and reabsorption of bile (also called resorption jaundice or *hepatogenous* jaundice, in contradistinction to *hæmatogenous*, which will be described hereafter), not only the coloring matter of the bile, but its other constituents, particularly the acids, are taken into the blood. As has been proved by numerous experiments, these acids possess to a peculiar degree the property of dissolving the red-blood corpuscles. By injecting weak solutions of them into the blood of animals, we may artificially induce the so-called hæmatogenous icterus, as the liberated coloring matter of the blood is transformed into biliary coloring matter. As it is firmly established, both that in biliary obstruction the bile-acids enter the blood, and that the absorption of these acids into the blood sets free the coloring matter of the latter and transforms it into biliary coloring matter, we may correctly say that every hepatogenous icterus is accompanied by a hæmatogenous, or, more accurately, every hepatogenous induces a hæmatogenous icterus.

Of the diseases of the liver that have already been described, some, such as fatty and lardaceous liver, never induce icterus, as they never cause compression of the bile-ducts; others, such as cirrhosis, cancer, and hydatids, sometimes induce biliary obstruction, sometimes they do not. Where the bile-ducts are compressed, if the retention of bile be only partial, its reabsorption and the icterus do not become excessive; the unimpeded flow of bile from the bile-ducts, which are not obstructed, gives some color to the fæces. The case is different when the ductus hepaticus or choledochus is compressed by tumors of the liver or plugged up by hydatids; then the biliary obstruction becomes absolute, the icterus very marked, and the fæces totally discolored.

Total retention of bile, with its results, is much more frequently caused by disease or compression of the excretory bile-ducts than by disease of the liver. This condition will occupy us in the next section; in this chapter we shall only speak of the changes induced in the liver by biliary obstruction, and of its results.

Anatomical Appearances.—The size of the liver may be increased by general excessive biliary obstruction, just as by decided congestion of the blood; but the swelling rapidly subsides as soon as the obstruction to the flow of bile is removed. The form of the liver is not altered by the enlargement. In high grades of the affection, the larger as well as the smaller bile-ducts appear dilated and distended with bile. The color of the liver is deep yellow, and, in the highest grades, olive green; it is not usually regular, but mottled. According to *Frerichs*, on microscopical examination, we sometimes find the entire contents of the liver-cells pale yellow, sometimes there is a deposit of fine granular pigment, particularly in the vicinity of the nuclei. After the disease has lasted some time, the liver-cells contain firm collections of pigment in the form of yellow, reddish-brown, or green rods, spheres, or angular fragments. The cells containing pigment lie chiefly in the vicinity of the central veins.

Even when the obstruction to the excretion of bile is not removed, the previously-enlarged liver may become smaller, and may even be reduced below its normal size; at the same time it acquires a dark-green or even black color, and loses its consistence, becoming soft and *capot*. In such cases the nutrition of the liver-cells has been impaired by compression of the afferent blood-vessels, and by pressure from the distended bile-ducts, perhaps also from the pressure of the bile collected in the cells. On microscopical examination, we find most of the cells broken down into a fine granular detritus, while some still contain pigment.

On autopsy of jaundiced bodies, collections of bile-pigment may be found in almost all the organs and fluids. Besides the characteristic color of the skin, conjunctiva, and urine (of which we shall speak more particularly when describing the objective symptoms of the disease), as soon as the body is opened we notice the lemon-color of the fat in the subcutaneous tissue, mesentery, pericardium, and elsewhere. The fibrinous coagula in the heart and blood-vessels, the fluid in the pericardium, and any pathological transudations or exudations of the pericardium, pleura, and peritonæum, have a distinctly jaundiced appearance. The less red the normal color of the different tissues is, the more marked is the pathological yellow color; hence it is more evident in the serous and fibrous membranes, the walls of the vessels, the bones, cartilages, etc., than in the muscles, spleen, etc. Only the brain, spinal marrow, and nerves form an exception, as only a slight color can be seen in them. *Frerichs* supports the previous observations, according to which the secretions proper, the saliva, tears, and mucus, contained no bile-pigment, while the albuminous and fibrinous exudations are rich in it. The changes in the kidneys, which were first fully described by the above observer, are very interesting. In old and intense cases of icterus he found the kidneys of an olive-green color, and some of the uriniferous tubules filled with a brown or black deposit. On more careful examination, in the pale uriniferous tubules, he saw the epithelial cells, which were really perfect, colored brown by pigment: the dark uriniferous tubules were filled with a coal-black, hard, brittle mass. The pigmentation of the epithelium began in the Malpighian capsule, increased in the convoluted tubuli uriniferi, while the black, coal-like masses were chiefly found in the straight tubules.

SYMPTOMS AND COURSE.—Premonitory symptoms almost always precede the characteristic signs of biliary obstruction. These consist of the symptoms of the disease that lead to contraction and closure of the bile-ducts; and, as this is most usually catarrh of the duodenum, they are most frequently those of gastro-duodenal catarrh. If these have existed for a longer or shorter time, the passage of the catarrh to the ductus choledochus, or the closure of the bile-ducts in any way, is almost always first shown by the peculiar dark color of the urine, and the light color of the fæces. But generally it is not these symptoms, but it is yellowness of the skin and eyes, that induces the patient to seek medical aid. Sometimes the skin is only slightly yellow, sometimes it is an intense saffron color; later, and in the higher grades, called melan-icterus, it may be greenish or even mahogany color. At those parts of the body where the epidermis is thin, so that the deeper layers of the rete Malpighii, where the pigment is located, shine through, the color is most intense, as on the forehead, alæ nasi, elbows, breast, etc. The yellow hue of the sclerotic, which may also be quite dark, is very characteristic of icterus, and is important in the diagnosis between jaundice and other discolorations. The yellow tint of the skin and sclera completely disappears by artificial light, so that we cannot recognize jaundice at night. We may see that the external mucous membranes are also yellow, by pressing the blood from the lips or gums of a patient with jaundice; when the finger is removed, the spot left will not be white, but yellow. Sometimes the urine is light brown, like thin beer, sometimes dark, like porter; after standing in the air, it almost always becomes greenish. If we agitate the discolored urine, its froth is distinctly yellow, and a strip of linen or white paper dipped in it becomes yellow, and this often suffices to distinguish between the coloring matter of the bile and other coloring matter in the urine. The test with nitric acid containing some nitrous acid is more certain. On adding this, the brown of the bile-coloring matter successively becomes green, blue, violet, red, and finally pale yellow. To note the changes well, we should carefully let some of the acid run down the inside of a champagne or test glass, containing the urine to be tested, so that it will reach the bottom, and there will be only a gradual admixture of the acid with the urine. If there be any bile-coloring matter present, and we let the urine stand a while, the various layers immediately above the nitric acid show different colors, and the above series of colors may be perfectly or partly distinguished from above downward. The reaction may be incomplete, or may fail entirely, if the urine has stood exposed to the air for some time, and already has a greenish color. According to *Frerichs*, the opposite occurs occasionally; the reaction does not take place till the urine has stood in the air for some time; until quite recently the occurrence of the bile-acids in the urine during icterus has been denied by celebrated authorities. There are certain difficulties in their detection in jaundiced urine by *Pettenkofer's* test, which shows very small quantities of the bile-acids by inducing a purplish-red color, when to the solution containing them we add a small amount of sugar, and then gradually add concentrated sulphuric acid. This reaction cannot be directly used when the fluid to be examined, as the urine, also contains substances which are directly colored by the addition of sulphuric acid. *Hoppe-Seyler* deserves the credit of having disproved the erroneous belief that in jaundice the urine contained only the coloring matter of the bile, and not the bile-acids, and of having proved the presence of the latter in the jaundiced urine by a complicated but perfectly reliable process.

Bile-pigment constantly occurs in the sweat also, so that the linen is colored yellow, particularly at those parts where the patients sweat much. The milk of nursing-women has also been found colored yellow.

The most noticeable change in the fæces, from obstruction to the flow of bile into the intestines, is their more or less complete discoloration. Where the excretory duct of the bile is incompletely closed, or the biliary obstruction partial, they have a loamy color, while they are clay-colored where the ductus hepaticus or choledochus is completely obstructed. As the amount of bile poured into the intestines during twenty-four hours is estimated at about two pounds, we may readily understand why the fæces are almost always dry. But, moreover, physiology teaches that, by excluding bile from the intestines, the absorption of fat is restricted, if not arrested; this explains the long-known fact that the fæces of jaundiced patients contain far more fat than do those of healthy persons. Professor *Trommer*, who examined the fæces of two of my students, who ate exactly the same amount of bread, butter, and cold meat, but of whom one was jaundiced, the other perfectly healthy, found far more fat in the fæces of the former than in those of the latter. Lastly, the putrid decomposition of the contents of the intestines appears to be avoided by the action of the bile on them; hence patients, in whom no bile is emptied into the bowels, usually suffer from flatulence, and the flatus passed, as well as the fæces, have a very disagreeable odor.

Besides the abnormal color of the skin, sclerotica, urine, sweat, milk, and besides the discoloration of the fæces, and the difficulties connected with the absence of bile from the intestines, we find that almost all patients with jaundice due to biliary obstruction rapidly *emaciate*, and become very *languid* and *sleepy*. As both the amylacea and protein substances are digested while there is no bile in the intestines, if there be no coincident gastric and intestinal catarrh, the emaciation can only be ascribed to the change in the absorption of fat. As has been previously stated, *Bischoff* has experimentally shown that a plentiful supply of fat may cause less consumption of the tissues of the body. It is very probable that the withdrawal of fat may have the opposite effect, and induce an increased use of the fat collected in the body. Even the exceptions, where jaundiced patients remain well nourished, although no bile reaches their intestines, do not disprove this explanation. For it has been observed that, while most dogs with artificial biliary fistula emaciate greatly, some remain well nourished, and it is particularly those that eat a great deal. In the same way, it has been observed that it is just those persons that have an excellent appetite and good digestion during their jaundice, who do not emaciate. Hence we may assume that failure of the supply of fat can be replaced by increased supply of hydrocarbons, and protein substances. The discovery, that in icterus the bile-acids are reabsorbed, and that their presence in the blood induces the disintegration of the red-blood corpuscles, shows another cause for the poverty of the blood, the emaciation and the cachexia, which often become very marked in protracted icterus. The languor, also, and the great inclination to sleep appear to depend not only on the bad nutrition, but to be greatly due to the toxic influence of the bile-acids on the nerves and muscles.

The *slow pulse* of patients with jaundice appeared to be intimately associated with the emaciation and loss of strength. It was not considered necessary to refer this symptom to the entrance of the constituents of the bile into the blood, and to compare their action to that of digitalis, for the retardation of the pulse was observed in the so-called hunger-cure, and in the convalescence from severe illness after the fever had stopped; but the useful experiments of *Röhrig* have shown that, in patients with icterus, the slow pulse mostly depends on the presence of bile-acids in the blood.

The same is true of the *itching* which annoys many patients with icterus. This has been referred to the dry, scaly state of the patients' skin, as it was also seen in senile marasmus. But its proportionate frequency in icterus, and its rare occurrence in marasmic conditions, render it probable that it is due to an irritation of the cutaneous nerves by the constituents of the bile deposited in the rete Malpighii. It is true, the itching is often absent in the highest grades of icterus, while in moderate grades it is very troublesome; and it is almost always periodical—peculiarities difficult to explain if the symptom be due to irritation from the acids or coloring matter of the bile.

*Yellow vision—Xanthopsia*—very rarely occurs in icterus. It is doubtful whether this be due to the yellow hue of the transparent media of the eye, or whether it depends on abnormal innervation, and is one of the first symptoms of acholia, of which we shall presently speak.

The course and termination of the disease depend chiefly on whether the obstruction to the evacuation of the bile can be removed soon, late, or not at all. In the first case, when the obstruction is overcome, the symptoms of retention of bile disappear quite rapidly, and the disease ends in recovery. At first, the fæces resume their normal hue, and the dark color of the urine and the symptoms due to saturation of the tissues, with pigmented nutritive fluid, soon pass off. The discoloration of the skin disappears last, particularly when the epidermis is thick. If enlargement of the liver has been observed while the flow of bile was obstructed, this also subsides when the flow becomes free. The strength and nutrition also improve rapidly.

Since, in icterus dependent on obstruction of the excretory gall-ducts, the return of the normal color to the fæces is almost always the first sign of improvement, the physician, and often also the patients, sually await its occurrence with great anxiety. Sometimes, however, the fæces acquire a weak, bilious color, without the bile-ducts having become pervious. This depends on their admixture with jaundiced blood-serum, or jaundiced inflammatory products. (Even in high grades of icterus the intestinal mucus contains no bile-coloring matter, otherwise the fæces would never lose their color.) Small hæmorrhages into the intestines, such as quite frequently occur during continued obstruction of the excretory bile-ducts, most commonly lead to error; they do so the more readily, as a very slight admixture of blood with the fæces does not induce any characteristic color, particularly when the blood-serum is jaundiced.

If the biliary obstruction continue for some time, or if it depend on causes that cannot be removed, the jaundice attains the highest grade, and the nutrition may suffer so much that the patient will finally die of marasmus or dropsy.

In rare cases the final result is hastened by the occurrence of *gastric* or *intestinal hæmorrhage.* These result from the same causes as in cirrhosis and pylephlebitis. The escape of blood from the vessels of the gastric and intestinal mucous membrane is just as much obstructed in the former case by the compression of the capillaries of the liver from the distended gall-ducts, as it is in the latter by the compression of the hepatic vessels from the contracting connective tissue, or by the obstruction of the portal vein. But in explaining these hæmorrhages, besides the mechanical obstruction to the flow of blood, we must also take into consideration the disturbed nutrition of the gastric and intestinal capillaries, especially as, in the course of icterus, hæmorrhages also occur in other parts, particularly in the skin, as petechiæ. We have repeatedly said that the inclination to bleeding, the so-called hæmorrhagic diathesis, can only be explained by disturbed nutrition of the walls of the vessels, and that the latter very frequently occurs in advanced anæmia and cachexia.

The occurrence of severe disturbances in the nervous system during an attack of jaundice is far more dangerous. These rarely begin with delirium or convulsions; they are rather apt to commence with paralytic symptoms. The patients are insuperably sleepy, finally become soporose, and die in this state. *Henoch* calls attention to the fact that *Hippocrates* had recognized the bad prognostic indication of this sign, for he says, *Ex morbo regio fatuitas aut stupiditas mala est.* The most varied hypotheses have been started about the occurrence of these brain- and nervous symptoms. Recently the inclination has been mostly toward the view that these symptoms depend on poisoning by the bile-acids, for, on injecting these into the blood of animals, we observe symptoms of poisoning indicative of paralysis of the nervous system. To this it is objected that the bile-acids exist in the blood in every case of icterus, while these severe nervous affections are very rare. *Leyden* attempts to meet this objection by calling attention to the fact that the severest symptoms are avoided by the continued excretion of the bile-acids through the kidneys, which prevents their excessive collection in the blood. The correctness of this explanation appears to be supported by the fact that a remarkable fatigue is scarcely ever absent, no matter how slight the jaundice, as well as the fact that a diminution of the secretion of urine has been always considered as having a bad effect on the course of jaundice. On the other hand, it is opposed by the fact that, in hæmatogenous icterus, in which, as *Leyden* very particularly notices, no bile-acids can be discovered in the urine, the same severe nervous difficulties are observed even more frequently than in hepatogenous icterus. I do not consider the question as settled. The proportionately frequent occurrence of severe disturbances of innervation in cases where there is only a slight amount of icterus, but a severe and extensive degeneration of the liver, may be far more readily explained by the hypothesis of *Frerichs*, already alluded to, than by the equally hypothetical poisoning by the bile-acids. Moreover, the assertion, that the excessive collection of bile-acids in the blood and in the tissues is avoided by their continued excretion through the kidneys, is opposed by the fact that the excessive collection of the bile-pigment is not prevented by its continued and plentiful excretion through those organs. If the affair were as simple as *Leyden* suggests, the frequency of the occurrence of symptoms of poisoning would still be in some proportion to the intensity of the icterus, which is not the case, as is well known.

In slight grades of biliary obstruction, physical examination does not show any enlargement of the liver. In higher grades, on the contrary, such as those due to complete closure of the ductus hepaticus or choledochus, we may often recognize very decided enlargement of the liver by palpation and percussion. Its surface appears smooth, and as the consistence is increased the lower border is very distinct. If the ductus choledochus be closed, we may generally feel the distended gall-bladder as well as the liver-tumor. If the liver-dulness diminish without decrease of the icterus, it is a bad sign, for it indicates consecutive atrophy of the liver.

TREATMENT.—We can only treat those cases of biliary obstruction successfully where we are in a position to fulfil the causal indications. Hence we are powerless against the cases caused by most hepatic diseases, such as echinococcus, carcinoma, or cirrhosis, while we are sometimes successful in treating those cases due to obstructions in the bile-ducts. The remedies vaunted as specifics in icterus are such as have a favorable effect on the diseases of the bile-ducts; we shall speak of them in the next section. This is particularly true of the Karlsbad springs, which have a world-wide reputation for their efficacy in jaundice. Many patients, who have gone to Karlsbad with the most intense jaundice, return cured in a few weeks; but these are only persons whose icterus depended on catarrh of the bile-ducts, or on their obstruction by gall-stones. If jaundiced patients, with an incurable obstruction of the bile-ducts, go to Karlsbad, their jaundice is not improved by the use of the waters; but they die sooner than they otherwise would, because the symptoms of congestion are increased, and the destruction of the liver-cells is hastened by the augmented secretion. This assertion is well supported by cases. The internal use of nitrate of potash, calomel, of the bitter and soluble extracts, of emetics and purgatives, does just as little good as the Karlsbad waters in icterus, unless it fulfil the causal indications.

When we succeed in removing the obstacle to the excretion of bile, the indications from the disease do not require any thing further; when we cannot succeed, they cannot be fulfilled.

The symptomatic indications require, first of all, an improvement of the depressed state of the patient by a proper diet. We should order meats, particularly cold meats and strong soups; but as fat is not absorbed when the bile does not enter the intestines, and consequently is not well borne, the use of gravies, butter, etc., should be just as strictly forbidden, while the patients remain at home, as when they go to Karlsbad, where, according to the diet list, the use of these articles is very reprehensible. In the next place, we should particularly attend to the constipation, from which most patients with jaundice suffer, and which depends partly on dryness of the fæces, partly on absence of irritation of the intestinal mucous membrane from the bile; but we should avoid saline laxatives, using instead slight drastics, such as infusion of senna, lenitive electuary, and rhubarb and aloes. As quantities of bile-pigment are evacuated with the urine, we may attempt to hasten the disappearance of the icterus by prescribing diuretics, such as bitartrate of potash, soluble cream of tartar, acetate and carbonate of potash. These are urgently indicated when the amount of urine is diminished, as the obstruction of the uriniferous tubules, to which *Frerichs* has called attention, may cause a retention of the constituents of the urine, and it is possible that the obstructions may be washed away by an increased secretion of urine. When the biliary obstruction has been removed, we may advise lukewarm baths, steam, soap-and-potash baths, to cause a more rapid removal of the epidermis, and thus relieve, as quickly as possible, the annoying itching and the jaundiced color.

## CHAPTER XIII.

### ICTERUS WITHOUT REABSORPTION OF BILE—HEMATOGENOUS ICTERUS.

ETIOLOGY.—Physicians have long observed that there are cases of jaundice that cannot be referred to retention and reabsorption of bile.

The supposition, that in severe and extensive degenerations of the liver the constituents of the bile collect in the blood, because they are not excreted by the affected organ, is completely refuted by the reliable observation that the bile and its constituents are not formed in the blood and simply excreted by the liver, but that the bile is first formed by the liver.

The attempt also to refer some cases of jaundice to *spasm* of the excretory bile-duct, because no obstruction to the excretion of the bile could be found on autopsy, may be regarded as a failure. As the excretory bile-ducts contain muscular elements, it is of course possible that they may be temporarily closed by spasmodic contraction, but it is excessively improbable that such a spasm would continue so long, as physiological experiments prove is necessary, as to induce the grade of biliary obstruction requisite for the passage of bile from the bile-ducts into the blood-vessels and lymphatics, and the production of icterus. The occurrence of icterus spasticus is more than doubtful.

Lastly, it is also very improbable that, under certain circumstances, more bile is prepared than the bile-ducts can accommodate, and that icterus is induced because part of the excess of bile enters the blood-vessels and lymphatics.

*Frerichs* has advanced a new hypothesis for the explanation of this form of jaundice: he considers it probable that a filtration of bile from the bile-ducts into the blood-vessels results not only from an overfilling of the bile-ducts, but from too little fulness of the blood-vessels. The icterus not unfrequently seen with thrombus of the portal vein is explained thus: blood is supplied to the liver only by the hepatic artery, and thus the hepatic vein and capillaries are not so well filled; some cases of icterus neonatorum are explained by the sudden cessation of the supply of blood to the liver through the umbilical vein, and the icterus in yellow fever because the roots of the portal vein are emptied by the large intestinal hæmorrhages.

The views regarding the occurrence of jaundice without retention and reabsorption of bile have totally changed since the observations of *Virchow*, *Kühne*, and *Hoppe-Seyler* have shown that bile-coloring matter may be formed from the free coloring matter of the blood without the action of the liver; and we may induce artificial jaundice in animals, by injecting substances that dissolve the blood-corpuscles.

There is now no doubt that some of the formerly enigmatical forms of icterus are due to the disintegration of blood-corpuscles, and the transformation of the freed coloring matter circulating in the blood into bile-coloring matter. This is particularly true of those cases of icterus occasionally caused by poisoning from chloroform or ether; for, as experiment proves, these substances possess the power of dissolving blood-corpuscles. The slight icterus in excessive hydræmia, to the explanation of which but little attention has been paid, also doubtless depends on the fact that blood-corpuscles are destroyed by a large amount of water in the blood.

This mode of origin is very probable, although not absolutely proved, for other varieties of jaundice—as in that occurring after snake-bites, in that observed constantly in yellow fever, quite often in recurrent fever, septicæmia, and puerperal fever, and more rarely in other infectious diseases, and acute diseases accompanied by severe fever. It is a very interesting fact that in the latter diseases even the older physicians suspected a *dissolutio sanguinis* when they ran a pernicious course, when the fever was very high, when there was great prostration, and when severe nervous symptoms, jaundice of the skin and conjunctiva, appeared. We will not discuss the question as to whether the disintegration of the blood-corpuscles in the above diseases is the result of the high temperature, or whether, in high fever, products form from the excessive transformation of tissue which dissolves the blood-corpuscles.

The jaundice in pylephlebitis also, and even some of the cases where it occurs with abscess of the liver, belong to the hæmatogenous form. A short time since I had the opportunity of observing a patient with a large abscess of the liver, who suffered from symptoms of jaundice as long as an intense fever, with very typical intermittent course and severe chills, lasted. When the fever ceased, the bile-coloring matter and the albumen that had occurred coincidently in the urine disappeared, and the jaundiced hue of the skin and conjunctiva was soon lost. The jaundice, which, as previously stated, occasionally occurs with ulcerative endocarditis, appears also to be hæmatogenous; but I think it very doubtful whether any of the cases of icterus neonatorum belong here.

Lastly, I must repeat my former opinion that every hepatogenous icterus, resulting from reabsorption of the bile-acids, leads to a hæmatogenous icterus.

ANATOMICAL APPEARANCES.—Jaundice that is not due to retention and reabsorption of bile rarely reaches a high grade; on autopsy we usually find only a faint yellow color of the outer coat of fat and of the other tissues. A more important point in the *post-mortem* diagnosis of hæmatogenous icterus, and for distinguishing it from the hepatogenous, is the circumstance that in the former the liver is not strongly jaundiced, while in the latter the signs in the liver of reabsorption of bile are always the most prominent. The normal color of the contents of the intestines, particularly the greenish hue of the contents of the duodenum, as well as the positive observation that the bile-ducts and excretory passages are quite pervious, leaves no doubt that it is a case of hæmatogenous icterus. But, in judging of the perviousness of the bile-ducts, we must guard against mistakes. The fact, that by pressing on the gall-bladder we may press a few drops of bile from the ductus choledochus into the duodenum, does not at all prove that during life this passage was perfectly free; on the contrary, this may be done where, from the presence of a gray plug of mucus and epithelium, in the intestinal portion of the ductus choledochus, we may be quite certain that there was catarrh of the duct, and a hepatogenous icterus. *Buhl* and *Liebermeister* have the credit of first calling attention to the changes found in the liver and other organs in hæmatogenous icterus. According to *Liebermeister*, the changes in the liver-cells and in the epithelium of the uriniferous tubules consist "in an excessive collection of small and fine fat globules, or else only of cloudy, probably albuminous substance in them;" in other cases, "the cells have fallen into detritus and numerous fat globules have appeared; analogous changes also occur in the substance of the heart." These *parenchymatous degenerations* (*Liebermeister*) of the liver, kidneys, and heart, show that, under the influence of the same injurious causes that induce a disintegration of the blood-corpuscles (that is, a parenchymatous degeneration of the blood), the firm tissues of the body may be affected in the same way.

Symptoms and Course.—As we have already shown, hæmatogenous icterus is only one symptom of extended disturbances. Hence we cannot well give an exact description of its symptoms and course, and we must confine ourselves to calling attention to those points which, occurring in icterus, show that it is not due to reabsorption of retained bile, but to a transformation of the coloring matter of the blood into that of the bile in the circulation.

Attention to the etiology aids us greatly. If icterus occurs after the action of the injurious influences mentioned in the first part of this chapter, or in the course of some of the diseases there mentioned, the presumption is that the case is one of hæmatogenous icterus. This supposition is strongly supported if the fæces appear of a normal or very dark color. If, with the commencement of the symptoms of jaundice, the beat of the heart and pulse become irregular and intermittent; if, along with the coloring matter of the bile, albumen appear in the urine; and if, besides these symptoms, we have severe disturbances of the nervous system, I consider the diagnosis of hæmatogenous icterus as quite certain. *Leyden* also reckons among its criteria the discoloration of the urine, which is very slight in proportion to that of the skin, and particularly the absence of bile-acids from the urine. If it were proved that, even in moderate grades of hepatogenous icterus, the bile-acids could be constantly and certainly found, their absence from the urine would, indeed, be a certain criterion of hæmatogenous icterus.

TREATMENT.—Hæmatogenous icterus does not require any particular treatment; it disappears as soon as we can remove the original disease. But, unfortunately, we are rather powerless in this respect; still, in those cases occurring during very intense fever, we may expect most from an antipyretic course of treatment.

## CHAPTER XIV.

### ACUTE YELLOW ATROPHY OF THE LIVER.

ETIOLOGY.—In acute yellow atrophy of the liver—a very obscure disease, for which we find no analogy—in a short time the liver becomes smaller, soft, and pulpy, and, on microscopical examination of the atrophied and softened organ, we find that the liver-cells are mostly destroyed.

From the fact that the liver, which has been swollen by continued biliary obstruction, occasionally becomes smaller, and softens, and that, in this form of atrophy, the liver-cells are also found disintegrated, acute yellow atrophy has been referred to obstruction of the bile in the finer bile-ducts. But this supposition is opposed, on the one hand, by the circumstance that, in acute yellow atrophy of the liver, the gall-ducts are found empty, or filled with mucus; and, secondly, that no obstruction to the flow of bile can be found in them.

Other observers take the view that the destruction of the liver-cells is, it is true, the result of pressure on them, or on the vessels nourishing them, and that this pressure is exercised by the distended bile-ducts, but that the over-filling and distention of the bile-ducts do not depend on stasis of their contents, but on excessive formation of bile, on *polycholia*. But since, neither at the commencement of acute yellow atrophy of the liver, nor during its first stages, are there signs of increased flow of bile into the intestine, the supposition of excessive formation of bile, as the cause of this disease, appears neither proved nor probable.

Most recent pathologists consider acute yellow atrophy of the liver as the result of a peculiar form of hepatitis; and, indeed, its acute course, and the rapid and extensive destruction of the liver-cells, would indicate an inflammatory process. Moreover, *Frerichs* claims to have found a free exudation surrounding the lobuli of the liver, in some parts of the organ, where the process was not yet far advanced. Leaving out of the question this interstitial exudation, which is probably not constant, acute yellow atrophy of the liver would appear to belong to the *parenchymatous inflammations*, i. e., to those forms of inflammation where there is no free exudation between the elements of the tissue, but where the elements of the parenchyma themselves swell by taking up an albuminous substance, and subsequently undergo a combined molecular and fatty degeneration. Against this view, which was advanced by *Liebermeister*, or, at least, first precisely stated by him, the most we can say is, that the course of inflammations in other organs, particularly in the kidneys, is entirely different, and that there is no parenchymatous inflammation in which, in a very short time, while the affected organ rapidly becomes smaller and softer, the tissue elements are destroyed, as they are in acute yellow atrophy of the liver.

Whether the destruction of the parenchyma-cells of the liver, in acute yellow atrophy, be of inflammatory origin or not, this disease is apparently not primary and idiopathic, but the result of a severe constitutional affection. The supposition that this constitutional disease is due to the action of a poisonous, miasmatic substance taken into the blood, to an infection, cannot at present be proved, although the occasional epidemic occurrence of the disease is favorable to such a view. The popular comparison of acute yellow atrophy of the liver with the fatty liver seen after poisoning by phosphorus, is unsound, and has caused many mistakes. In phosphorus-poisoning we have a fatty *infiltration;* in acute yellow atrophy there is a fatty *degeneration* of the liver-cells: these are decidedly different forms of disease. I consider the attempt to refer acute yellow atrophy of the liver to poisoning by the bile-acids as a failure. Even the slight grade of the icterus existing in most cases refutes the correctness of this hypothesis. It must be acknowledged that the greater or less intensity of the jaundice gives the best means of judging whether much or little bile has been reabsorbed; for, although we can only determine from this the amount of bile-*coloring matter* that has been reabsorbed, still this gives the means for deciding the amount of *bile-acids* absorbed. It is true, *Leyden* has attempted to explain the frequent absence of symptoms of poisoning by the bile-acids in excessive and protracted icterus, by saying that, in most cases, their collection in the blood is avoided by their elimination through the kidneys. But this explanation is very doubtful. Daily experience teaches that, in closure of the excretory bile-ducts, in spite of large quantities of bile-pigment being steadily thrown out by the kidneys, the icterus increases regularly as long as the bile-ducts remain closed. What right have we to suppose that absorption of the bile *acids* into the blood is compensated for by their excretion through the kidneys, when it is so evident that this does not occur in the case of the *coloring* matter?

The disease is very rare; it never occurs in childhood. It is met with more frequently in women than in men, and most frequently during pregnancy. It is interesting to note that pregnancy, which greatly favors the occurrence of parenchymatous inflammation of the kidneys, is undoubtedly accompanied by a predisposition to analogous disease of the liver.

ANATOMICAL APPEARANCES.—In high grades of acute yellow atrophy of the liver, the organ is much diminished in bulk, occasionally being less than half its normal size. Its thickness is particularly diminished, so that it appears flattened. Its serous covering is loose, often even in folds. The parenchyma is relaxed and flabby, and the liver is sunk in against the posterior wall of the abdomen. The organ is dull yellow, its consistence diminished, and its acini irrecognizable. On microscopical examination, instead of the normal liver-cells, we only find detritus masses, fat globules, and pigment granules. In the right lobe, where the changes were less advanced, *Frerichs* found "between the lobules, surrounded by hyperæmic vessels, a dirty grayish-yellow mass, which separated them. Farther off the hyperæmia of the capillaries disappeared, the lobules became smaller and yellower, and the intervening gray substance was in excess." The gall-ducts and bladder usually contain a scanty mucous secretion. The fæces are generally only slightly colored; the contents of the intestines are often bloody. The spleen is enlarged in most cases. Ecchymoses are frequent, particularly in the peritonæum and in the gastric and intestinal mucous membrane, and not very rarely in the other serous membranes and in the skin. In the kidneys, *Frerichs* not only found deposits of pigment in the epithelium, but also fatty degeneration and disintegration of the epithelial cells. The same observer found quantities of *leucin* in the blood, and in the urine evacuated from the bladder; in the latter there were also *tyrosin* and a peculiar extractive matter.

SYMPTOMS AND COURSE.—The *first stage* of the disease has no very characteristic symptoms. The patients suffer from loss of appetite, pressure and fulness in the epigastrium, and other symptoms that remind us of gastro-intestinal catarrh. There is also in most, but not in all, cases a moderate jaundice, which may excite the suspicion that the catarrh of the duodenum has invaded the ductus choledochus. Not a single symptom betrays the great danger overhanging the patient.

In the *second stage* the jaundice increases, the region of the liver becomes sensitive to pressure, the patients complain of severe headache, become restless, excited, and finally delirious. Occasionally the excitement extends to the motor nerves, so that there are local or general muscular twitchings. But soon, and sometimes without any previous symptoms of irritation, the patient is seized with insuperable depression and lassitude; he falls into a deep sleep, from which he can at first be aroused momentarily, particularly by pressure over the liver, but later cannot be awakened at all. Then the previously normal or even retarded pulse almost always becomes frequent. The temperature rises very high; tongue and gums become dry and covered with sordes; the fæces and urine are evacuated involuntarily. The collapse increases, the very frequent pulse becomes smaller, there is copious perspiration, and, without arousing from his coma, the patient usually dies the second day, more rarely the fourth or fifth day, or later. We might be tempted to regard the icterus in acute yellow atrophy of the liver as hæmatogenous, and to explain it by saying that the blood-corpuscles are destroyed in the same way as the liver-cells by the exciting cause of the disease. But the discoloration of the contents of the intestines, although incomplete, and the jaundiced appearance of the liver, as compared with the other organs, render the correctness of this explanation very questionable. The intensely bilious color of the liver shows that the discoloration of the contents of the intestines is not due to arrested production of bile, that is, to acholia. Nor can the icterus be referred to catarrh of the excretory bile-ducts, of which the first symptoms of the disease remind us, for neither the gall-bladder nor ducts are distended with bile. I agree with *Buhl* and *Bamberger*, who refer the obstruction and reabsorption of bile to a blocking up of the origin of the bile-ducts by fatty and molecular detritus of the liver-cells. It is difficult to explain the *brain-symptoms*, which are the most prominent symptoms of acute yellow atrophy of the liver. We have already spoken of our reasons for not referring them to poisoning by the bile-acids. *Bamberger* also says: "So much is certain, the brain-symptoms cannot be regarded as cholæmic, for both the grade and duration of the jaundice are too slight for this to be the case." Since in acute yellow atrophy the liver-cells are extensively destroyed, and as in this disease abnormal products of destructive assimilation have been found in the urine, there is some ground for attributing the brain-symptoms in acholia to poisoning by noxious substances, which are formed instead of the normal products of the change of tissue. But I do not consider even this explanation as proved. It is possible that the same cause may induce both the degeneration of the liver and the brain troubles. The hæmorrhages occurring during the disease are apparently the result of disturbed nutrition of the capillary walls, of an acute hæmorrhagic diathesis, which is also seen to occur in many other severe diseases that affect the composition of the blood. *Frerichs* refers the intestinal hæmorrhage, and the enlargement of the spleen, partly to compression of the capillaries of the liver.

Physical examination gives very important results, as it shows very rapid decrease of the liver-dulness, the sole pathognomonic symptom of acute yellow atrophy of the liver. At first the percussion-sound becomes very full in the epigastrium, because the diminution in size begins in the left lobe of the liver; but often, even after a few days, we can find no trace of liver-dulness. The total disappearance of liver-dulness is partly due to the relaxed organ shrinking together and being pressed against the spine by the inflated intestines. Besides the decrease of liver-dulness, the increase of spleen-dulness is important; but the enlargement of the spleen is not always so marked that it can be made out by physical examination, and sometimes it does not occur.

TREATMENT.—It is evident that we can say nothing that is reliable and founded on experience, concerning the treatment of a disease of which it is doubtful whether it ever ends in recovery. If we could distinguish the first stage of this disease from that of catarrhal jaundice, we should apply leeches about the anus, cold compresses over the right hypochondrium, and give saline laxatives. In the second stage, according to all observations made, abstraction of blood has an injurious effect on the course of the disease. On the other hand, powerful drastics, aloes, extract of colocynth, croton-oil, etc., are recommended, particularly by English physicians. While there are symptoms of irritation in the nervous system, great excitement, delirium and subsultus, ice is usually applied to the head; when paralysis occurs, cold douches are given; this treatment, having been found of occasional benefit in inflammatory brain affections, has been applied to the cases from poisoning. Although almost all comatose patients revive momentarily during the douche, we cannot count on a permanent benefit from it in acute yellow atrophy of the liver. The same is true of the internal and external use of irritants, which are recommended for the paralytic symptoms; of the mineral acids given when petechiæ occur, and of the ice-pills that are prescribed for the severe vomiting, and for the gastric and intestinal hæmorrhage.

# SECTION II.

## *DISEASES OF THE GALL-DUCTS.*

### CHAPTER I.

#### CATARRH OF THE GALL-DUCTS—ICTERUS CATARRHALIS.

ETIOLOGY.—The larger bile-ducts of the liver, the ductus hepaticus, cysticus, choledochus, and the gall-bladder, are lined with a mucous membrane, having cylindrical epithelium and racemose glands. This, like other mucous membranes of similar texture, is quite often the seat of catarrhal inflammation. The small calibre of the gall-ducts and excretory passages gives peculiar importance to this otherwise mild disease. The narrow canals are easily obstructed by the swelling of their mucous membrane, and by collections of mucus, and these are the most frequent causes of obstruction and reabsorption of bile.

In some cases catarrh of the bile-ducts results from excessive hyperæmia of the liver, in which the mucous membrane of the bile-ducts participates. Thus hyperæmia of the parenchyma of the liver and of the gall-ducts accompanies the development of cancer of the liver. If this reach a high grade, it may lead to catarrh of the bile-ducts, and thus to icterus. Too little attention has hitherto been paid to this mode of origin of icterus in carcinoma of the liver. Cases of carcinoma of the liver occur in which there is not the slightest doubt that the icterus depends on this catarrh of the bile-ducts. If the jaundice be only temporary, and the fæces are more or less discolored as long as the jaundice lasts, and are again normally colored when it disappears, we cannot refer the jaundice to compression of the bile-ducts or excretory passages by a cancerous tumor, but must refer it to a cause that comes and goes, or at least increases and diminishes. We have such a cause in the hyperæmia which is present in all organs in the vicinity of new formations, particularly of carcinoma, and which occasionally becomes excessive, and at other times diminishes. The same is true of the *temporary* icterus in multilocular echinococci, and in many of the cases accompanying heart-disease, emphysema of the lungs, and other diseases which impede the escape of blood from the liver. We shall speak in a separate chapter of the intense catarrh caused by gall-stones, as they readily induce ulceration, and then lead to severe and peculiar symptoms. Lastly, perhaps the irritation from abnormal bile may induce catarrh of the bile-ducts; but this has never been proved, and is very problematical.

By far the most frequent cause of catarrh of the bile-passages is the propagation of the catarrhal inflammation about the opening of the ductus choledochus into the duodenum. This duodenal catarrh is almost always accompanied by gastric catarrh, and hence the jaundice caused by it is usually called gastro-duodenal jaundice, or icterus simplex, on account of its frequency, freedom from danger, and its mild course. The gastric and duodenal catarrh which extends to the gall-ducts may arise from the most various causes, and for the etiology of gastro-duodenal jaundice we refer to what has been said of the etiology of gastric and intestinal catarrh.

Anatomical Appearances.—In acute catarrhal inflammation the mucous membrane of the gall-ducts is reddened, relaxed, and swollen. Its surface is covered with mucous and epithelial masses. If the swelling of the mucous membrane be considerable, the ductus choledochus becomes impassable, particularly that part which traverses the wall of the duodenum transversely, running for some lines between the layers of the wall (the "portio intestinalis"), while the bile-ducts in the liver are dilated, and filled with bile containing more or less mucus. The parenchyma of the liver also shows the previously-described characters of moderate biliary obstruction. After the catarrh has lasted some time, the redness of the mucous membrane subsides, but its swelling and hypertrophy, together with a plug of mucus and epithelium, form an insuperable obstacle to the flow of bile. In such cases the bile-ducts are often enormously dilated, and the enlarged liver shows the signs of great biliary obstruction. The dilatation and distention with bile often commence in the ductus choledochus immediately above the obstructed portio intestinalis. (For the state of the gall-bladder in contraction or closure of the excretory bile-ducts, see Chapter III.)

Symptoms and Course.—In most cases catarrh of the bile-ducts is readily recognized by the symptoms of obstruction and reabsorption of bile. When these appear gradually, and increase slowly, our suspicions should first be directed toward catarrh of the bile-ducts, because catarrhal jaundice is so frequent, as compared with other forms of the disease. But this is scarcely ever a primary affection; it almost always accompanies catarrh of the gastric and intestinal mucous membrane; hence it is almost characteristic of catarrh of the bile-ducts, that symptoms of gastric and intestinal catarrh precede the jaundice for days, or even weeks, and continue while the disease lasts. In this sense, we may consider the coated tongue, bad taste, eructations, and other dyspeptic symptoms, as among the premonitions and symptoms of catarrhal jaundice. The longer the catarrh of the bile-ducts continues, and the more completely the excretory duct is closed by it, the greater will be the discoloration of the fæces, and the more intense the jaundiced color of the skin and urine, and the more the general health and nutrition of the patient will suffer. The liver appears distinctly swollen in many cases, and in some of them it is considerably enlarged.

If the disease run a favorable course, the improvement shows itself in a week or two, by a return of appetite, by the tongue cleaning off, and by decrease of the dyspeptic symptoms. Then we may hope that the catarrh of the bile-ducts will subside with the gastro-duodenal catarrh, and in fact, after a few days, the returning color of the fæces shows that the ductus choledochus is open, and the clearer color of the urine indicates that the biliary obstruction is less, and that less bile is reabsorbed. The bile-pigment deposited in the rete Malpighii disappears more slowly. After the fæces are strongly colored with bile, and the urine has regained its normal tint, the skin remains jaundiced for a time, and finally this last symptom of the disease disappears. In other cases the catarrh of the bile-ducts, as well as that of the stomach and duodenum, becomes chronic. The disease drags on for weeks or months, the jaundice becomes excessive, the patients emaciate decidedly, and the liver enlarges considerably. But these cases also almost always terminate in recovery, particularly under proper and energetic treatment; and the biliary obstruction caused by catarrh of the ducts very rarely runs an unfavorable course with the symptoms before described.

TREATMENT.—Experience teaches that catarrh of the bile-ducts rapidly disappears when the catarrh of the intestinal mucous membrane, which has attacked the gall-ducts, subsides. Hence the *causal indications* require the same measures that we have recommended in the treatment of gastric and intestinal catarrh. Under the circumstances there mentioned, an emetic may be indicated; in other cases diaphoretics, in still others, careful regulation of the diet suffices. We shall not repeat in detail what we have previously said, but only call attention again to the excellent effect in gastric and intestinal catarrh of the carbonates of the alkalies, particularly as existing in the Karlsbad and Marienbad waters. While we have denied all *direct* influence of the Karlsbad waters on biliary obstruction and jaundice, still there is no better treatment for most patients than the use of these waters, because in the majority of cases none better fulfils the causal indications. If the circumstances of the patient do not allow of his being sent to a watering-place, we may let him use the "Karlsbad diet" at home, and order soda-water or artificial Marienbad or Karlsbad water. From this treatment alone we shall see the most favorable and speedy results in catarrhal jaundice.

In some cases the *indications from the disease* may be fulfilled by the administration of an emetic. During the act of vomiting, the bile is forced out of the gall-ducts and bladder, toward the mouth of the ductus choledochus, and an obstructing plug of mucus may thus be pressed out of the latter. We might give emetics far more frequently, if the obstruction of the ductus choledochus were not more frequently caused by the swelling of the mucous membrane than by mucous plugs, and if we did not fear that the untimely use of an emetic might render worse the gastro-intestinal catarrh. Nitro-muriatic acid has a great reputation in the treatment of catarrhal jaundice; it is used externally, in the form of foot-baths (℥ ss—j. to a foot-bath) and as fomentations over the liver, or internally (ʒ ss—j. to ℥ vj. of mucilage, a tablespoonful every two hours). The internal administration might possibly have a favorable influence on the intestinal catarrh; perhaps it might also excite contractions in the excretory bile-ducts, and thus cause the expulsion of obstructing coagula. The external use of aqua regia would scarcely be of any benefit. The action of the drastics is explained by the effect they have of increasing the peristaltic action of the intestines, which extends to the ductus choledochus. But generally these have no favorable influence on the catarrhal jaundice, for which they are given so much. The exhibition of calomel (gr. j every evening), and of the Vienna decoction (two tablespoonfuls every morning), according to the so-called English method, is objectionable, although many patients with catarrhal jaundice recover in spite of this treatment. Slight laxatives are only advisable when there is obstinate constipation. Then we may use some one of the tartrates, particularly tartrate of potash, or a decoction of tamarinds (℥ j—ij to ℥ vj—viij) with acid. tartar. (℈ j—ʒ ss.) and syrup. sennæ c. mannæ; or we may prescribe infusum sennæ compositum or lenitive electuary.

## CHAPTER II.

### CROUPOUS AND DIPHTHERITIC INFLAMMATION OF THE GALL-DUCTS.

INFLAMMATIONS of the gall-ducts with fibrinous exudations are exceedingly rare, and when they do occur it is only in the course of severe diseases, such as protracted typhus, septicæmia, cholera, etc.

In *croupous* inflammation, we find the mucous membrane of the gall-bladder covered with a more or less firm false membrane, and in the ducts we find tubular coagulations, enclosing inspissated bile, and causing biliary obstruction. In *diphtheritic* inflammation, the tissue of the mucous membrane is infiltrated at certain places with a fibrinous exudation, which causes sloughing of the mucous membrane, and from separation of the slough deep losses of substance occur. These processes cannot be recognized during life. Even an intense icterus occurring during typhus, septicæmia, cholera, etc., cannot be referred to a croupous or diphtheritic inflammation of the bile-ducts, as it far more frequently occurs without any perceptible change in them.

## CHAPTER III.

### OBSTRUCTION AND CLOSURE OF THE EXCRETORY GALL-DUCT AND CONSECUTIVE DILATATION OF THE BILE-DUCTS.

Etiology.—The excretory gall-ducts are most frequently contracted and closed by catarrhal swelling of their mucous membrane and by collection of mucus. Among the further causes of this contraction and closure, and of consecutive dilatation of the bile-ducts above the contraction, are: 1. *Tumors* pressing on the excretory ducts, or growing into them. Sometimes they are caused by carcinoma of the liver, pancreas, stomach, or duodenum; sometimes by caseous or other degeneration of the lymphatic glands; sometimes by abscesses; rarely by hydatid cysts, aneurisms, or collections of hard fæces in the colon; and lastly, in a few cases, by multilocular echinococci, that have wandered into the gall-ducts or broken through into them, and have thence reached the ductus hepaticus. 2. Occasionally there is a more or less complete closure of the ductus choledochus, hepaticus, or cysticus, from *cicatricial contractions*, which remain in the excretory bile-ducts, or in the duodenum after ulcers have healed, or the thickening and consecutive atrophy of the peritonæum after peritonitis, particularly when the excretory bile-ducts are at the same time distorted or bent. 3. Lastly, *foreign bodies*, particularly stony concretions, obstruct or close the excretory bile-ducts.

When the ductus hepaticus is constricted or closed, the *consecutive dilatation* of the gall-passages is limited to the bile-ducts of the liver. But if the ductus choledochus becomes impervious, the ductus hepaticus, cysticus, and the gall-bladder, are all dilated. Finally, if the ductus cysticus alone be closed, no bile can enter the gall-bladder, it is true, but its mucous membrane continues to secrete mucus, and the gall-bladder becomes more and more distended by the secretion. This state is called *hydrops vesicæ felleæ*.

Anatomical Appearances.—According as one or other of the above causes prevails, the anatomical appearances vary so greatly, except in the general effect of the constriction or closure of the excretory bile-ducts, that we shall refrain from giving a detailed description of them. If its mouth be entirely closed, the ductus choledochus may attain the size of the small intestine, and the dilatation extends through the ductus hepaticus and its branches to the capillary gall-ducts. The gall-bladder is also dilated, but its dilatation is not in proportion to that of the gall-ducts, because, from the acute angle at which it opens, it is compressed by the dilating ductus choledochus. The liver shows the changes that we have described as characteristic of the highest grade of biliary obstruction; it is at first enlarged, and on section the dilated gall-ducts look like large cysts filled with bile; subsequently, they may become smaller, from atrophy of the liver. In *hydrops vesicæ felleæ*, the gall-bladder becomes a translucent, tense cyst, as large as a fist, or a child's head even, which contains a serous fluid, resembling synovia. Its muscular filaments are separated and atrophied, the mucous membrane has lost its structure, and has acquired the look of a serous membrane. In some cases closure of the cystic duct leads to atrophy of the gall-bladder; its mucous and bilious contents become inspissated, and changed to a chalky mass, while the walls are thickened and atrophied by chronic inflammation. Finally, there remains only a hard tumor, as large as a pigeon's egg, filled with a chalky pulp.

Symptoms and Course.—Contraction and closure of the ductus hepaticus and choledochus are characterized by the symptoms of excessive biliary obstruction, uncomplicated by those of gastro-duodenal catarrh, but occasionally accompanied by those of neoplasia or other growths in the abdomen, or of chronic peritonitis, or of gall-stones, etc. The icterus is more decided, and the fæces more discolored than in any other form of biliary obstruction. We usually find the liver enlarged, and if the ductus choledochus be closed we also feel the full and distended gall-bladder. Later the consecutive dilatation of the liver may also be observed. If we find cancerous tumors in the abdomen, if there has been colic from gall-stones, or if any other symptoms indicate the variety of the closure, the diagnosis becomes more certain. In most cases it is only possible to recognize the closure, without being able to make out its cause.

*Hydrops vesicæ felleæ* is readily diagnosed, if it be simple and not complicated with obstruction and closure of the ductus hepaticus or choledochus. If we find a pear-shaped, movable, occasionally fluctuating tumor starting from the incisura vesicæ felleæ, in a patient who is not jaundiced, we may diagnose closure of the cystic duct and distention of the gall-bladder by mucous secretion, or hydrops vesicæ felleæ.

As it is almost always impossible to remove the cause of the contraction or closure of the bile-ducts, it is impossible to treat them successfully.

## CHAPTER IV.

### GALL-STONES AND THEIR CONSEQUENCES—CHOLELITHIASIS.

Etiology.—In spite of numerous works on this subject, the origin of gall-stones is very obscure. Particles of mucus, or (far more rarely) foreign bodies in the gall-passages, appear to play an important part in the formation of gall-stones, for they form the nucleus in almost all cases, and they at least form the point on which the solid constituents of the bile may be deposited. It cannot be decided whether such deposits occur when the bile is normal, or only when it is somewhat concentrated, or when of abnormal composition. As a combination of chalk with bile-pigment is almost always deposited immediately around the above-mentioned nucleus, and as this combination almost always occurs in greater or less quantities, it is thought that excess of chalk in the bile, from drinking lime-water, has something to do with the formation of gall-stones. But, besides this, it is probable that the bile from which gall-stones, rich in cholesterin, are formed, has only a slight solvent power over cholesterin; and as it has been found that both cholesterin and biliary coloring matter with lime are dissolved by tauro-cholic acid and tauro-cholate of soda, it was very natural to consider a lack of tauro-cholic acid in the bile, or a decomposition of the tauro-cholic acid in the gall-bladder, as the possible cause of gall-stones.

Gall-stones occur more frequently in women than in men, far more frequently in old than in young persons; and, without our being able to explain why, they are particularly frequent in patients with carcinoma of the stomach or liver. Perhaps this is somewhat due to the catarrh of the bile-passages, which, as we said in Chapter I., often accompanies carcinoma of the liver.

Anatomical Appearances.—The size of gall-stones varies from that of a hemp-seed to that of a hen's egg. The smallest concrements are distinguished from gall-stones proper by the name of "biliary sediment." Most frequently there is only one calculus, in other cases there are a great number of them. Solitary stones are usually round or egg-shaped, or they have exactly the shape of the gall-bladder. Their surface is sometimes smooth, sometimes more rough and glandular. If there are several stones, they have almost always become smoothed on the sides that touch, and have a polyhedral form, with edges, corners, and smooth surfaces, or they have convex and concave facettes, which give them a peculiar appearance. Biliary calculi have a low specific gravity; when recently removed, they may be mashed by the fingers; when dried, they usually split, and finally break down into dust. Biliary calculi vary greatly in color; some are whitish, or pale yellow, from slight imbibition of bile, others are dark brown, and others still are greenish or blackish. They often consist of various layers, and light-colored strata may alternate with dark ones. Calculi, consisting chiefly of cholesterin, have a marked, striated crystalline structure, while those that contain chiefly biliary coloring matter with lime have an earthy fracture. Most biliary calculi are chemically composed of cholesterin, and have a small amount of biliverdine only about their nucleus; others consist of a mixture of cholesterin and biliverdine; the latter is sometimes distributed regularly through the mass, sometimes there are alternate layers of cholesterin and of the coloring matter of the bile with lime. We rarely meet calculi containing no cholesterin, but composed of bile-pigment and lime, or of carbonate and phosphate of lime (*Lehmann*).

In most cases no structural changes can be discovered in the coats of the gall-bladder, even when it contains numerous calculi with sharp edges. But occasionally, particularly in the fundus of the bladder, we find a considerable injection and puffing of the mucous membrane, or there is an ulcerative loss of substance of variable size and depth. The ulceration may lead to perforation of the gall-bladder. If this occurs before the wall has become adherent to neighboring parts, the contents enter the peritonæum and cause general peritonitis. If, on the other hand, the gall-bladder be perforated after it has formed firm adhesions with neighboring parts, there may be communication with the intestines or perforation outwardly. In some cases the inflammation induced by gall-stones is less destructive. The walls of the bladder are thickened, and, after a while, undergo cicatricial retraction; the contents become dry and chalky. And, finally, in such cases we find the biliary calculi embedded in a chalky mass, and firmly enclosed by the shrunken and atrophied gall-bladder.

In the gall-ducts of the liver, biliary calculi may excite suppurative hepatitis. In the excretory ducts, large calculi either cause ulceration and perforation or complete closure of the duct, so that the states described in Chapter III., excessive biliary congestion, or, if the cystic-duct be closed, hydrops vesicæ felleæ result. In some cases the bile-ducts are so dilated, by the pressure of the bile from behind, that the latter squeezes past the calculi, or that even comparatively large calculi are forced into the duodenum.

Symptoms and Course.—On autopsy, large calculi or numerous small concrements are often found in the gall-bladder, which apparently had no effect on the health of the patient during life. We may even say that it is exceptional for calculi in the gall-bladder to cause trouble, or betray themselves by any definite symptoms. Calculi of small size may even pass through the ductus cysticus or choledochus without exciting pain or any other symptom. The experiences at bathing-places, such as Karlsbad, where the stools are carefully examined for gall-stones, afford numerous examples of this.

Among the morbid processes which gall-stones induce, as above stated, we have already described suppurative hepatitis and obstruction of the excretory bile-ducts, so that we may here limit ourselves to describing the symptoms that arise during the passage of large gall-stones through the excretory passages, and, during their impaction there, the so-called gall-stone colic, as well as the symptoms of inflammation and ulceration of the gall-bladder and excretory ducts, which are excited by gall-stones in some few cases.

Gall-stone colic begins unexpectedly and suddenly at the moment a concrement passes from the gall-bladder to the ductus cysticus and becomes impacted there. The patient is seized with a piercing or griping, insupportable pain, which starts from the right hypochondrium and spreads over the whole abdomen, often also to the right side of the thorax, and even to the right shoulder. The abdominal muscles are cramped and very sensitive to pressure; the patients sigh and moan, double up, and roll about on the bed or floor. During all this there is no fever, but there are a number of other symptoms. The pulse becomes small, the skin cool, the face pale and distorted; occasionally the patient actually faints. In some cases the patients are affected with spasmodic trembling or chills; in other cases there are convulsions either general or limited to the right half of the body. There is most frequently obstinate sympathetic vomiting. After a few hours, or in severe cases not till next day, the sufferings of the patient are usually moderated, and the general disturbance quieted. This remission, during which, however, the patient still suffers severely, and still has a small pulse, and pale, cool skin, appears to correspond to the passage of the concrement from the cystic duct into the ductus choledochus, which, except the portio intestinalis, is a somewhat wider canal. The symptoms do not change very much till the concrement has passed the ductus choledochus and entered the duodenum. Then the patient is entirely free from pain and uneasiness; the pulse rises, the warmth of the skin returns, and the distortion of the countenance disappears. This passage, from great agony to complete ease, often occurs in a very short time, and then the contrast is very striking. In other cases the pain does not cease suddenly, but subsides gradually; this is probably because the irritated nerves of the gall-ducts are only gradually quieted, just as, when the eye is irritated by a foreign body, it remains irritable for a time after the body has been removed. Very rarely gall-stone colic terminates fatally with the symptoms of excessive faintness, passing into true palsy. Somewhat more frequently it is followed by the symptoms of permanent closure, or of inflammation and ulceration of the excretory bile-ducts.

Jaundice is not by any means a constant symptom of gall-stone colic. There can be no obstruction and reabsorption of bile from impaction of the calculus in the cystic duct; and, as we have before stated, even a temporary closure of the ductus choledochus does not result in icterus. Usually, after the symptoms of impaction have subsided, there is a slight jaundice, which is very temporary if the impaction in the ductus choledochus has not continued long. After reaching the duodenum, the calculi are rarely vomited; far more frequently they are passed at stool, and this passage is only exceptionally accompanied by abdominal pain or muco-bloody diarrhœa. It is almost always unnoticed, so that the calculus is only discovered on careful examination of the fæces. After the attack has passed, we are often unable to find any calculus, even if we place the fæces on a sieve and wash them through it. In such cases the probabilities are that the impacted concrements have gone back from the cystic duct into the gall-bladder.

There are still some dark points in our knowledge of gall-stone colic. It is remarkable and unexplained that, in some persons, gall-stones do not show any inclination to leave their place in the gall-bladder, while in others they very frequently pass through the ducts. We are not even clear as to how the calculi are pressed from the gall-bladder into the cystic duct, although it is most probable that they are floated onward, as it were, by the bile which is driven forward by the contractions of the gall-bladder. This view is supported, among other things, by the fact that gall-stone colic is particularly liable to occur during digestion. Lastly, we would suppose that, during a gall-stone colic, we might, from a variation in the intensity of the symptoms, distinguish three periods, the first corresponding to the impaction of the calculus in the very narrow cystic duct, the second to its passage into the somewhat wider ductus choledochus, the third to its impaction again in the very narrow portio intestinalis of the ductus choledochus; but we usually observe nothing that can be referred to these phases.

The inflammations and ulcerations caused by gall-stones do not induce any uneasiness till the peritonæum participates in the inflammation; then we have the above-described symptoms of partial chronic, and, occasionally, of acute peritonitis. The seat of the pain over the gall-bladder, as well as attacks of gall-stone colic that have preceded the pain, and, in rare cases, the discovery of a distention and fulness of the gall-bladder by calculi (*Oppolzer*), may excite the suspicion that the walls of the gall-bladder are inflamed and ulcerated on account of the concrements in it. If the gall-bladder be perforated before it has become adherent to the surroundings, we have the symptoms so often described, which are almost pathognomonic of the entrance of foreign bodies into the peritonæum, and, in a few days, the patient dies of diffuse peritonitis. If the neighboring organs have become adherent to the gall-bladder, when this is perforated, the pain is limited to the region of the gall-bladder; besides the above symptoms, there is disturbance of the functions of the bowels, and the symptoms of the disease often remain obscure till they are explained by the passage of a calculus, which is so large that it could not possibly have passed the ductus choledochus. Concrements entering the intestines through an abnormal communication between them and the gall-bladder may be so large as to pass through the bowels with difficulty, and may give rise to the symptoms of obstruction of the intestines. I have a cholesterin calculus larger than a pigeon's egg, which was given to me as an intestinal calculus, and which was passed with great pain by a lady, "after repeated attacks of hepatitis." If the inflamed bladder adhere to the anterior wall of the abdomen, we may occasionally feel it as a hard circumscribed tumor; subsequently the abdominal walls themselves become inflamed, an abscess forms in them, from which pus, bile, and often a great number of gall-stones, may be evacuated. The abscess does not always open at the part of the wall lying over the gall-bladder, but occasionally opens at a distance, after fistulous passages have formed in the walls. The fistula rarely closes after one or a few concrements have been evacuated; it more frequently lasts for a long while or always, constantly or at intervals pouring out bile, and, if the cystic duct be closed, a limpid fluid.

Inflammation and ulceration of the excretory bile-ducts, caused by calculi, are preceded by the symptoms of gall-stone colic; but this, instead of terminating, as it might, in complete recovery, leaves pain in the region of the liver, and great sensitiveness to pressure. When the impaction of the calculus, and the consequent inflammation, has affected the ductus choledochus, there is also intense icterus, and other symptoms of excessive biliary obstruction. Far more rarely, from its angular form, the calculus closes the excretory duct incompletely, so that small quantities of bile continue to reach the intestine. In such cases the fæces are not entirely discolored, and the icterus does not become so intense. In this case, also, there may finally be perforation, and consequent peritonitis (*Andral*). But more frequently, the patients die of the results of biliary obstruction, with the symptoms of marasmus, or of acholia.

TREATMENT.—We should try to preserve patients, who have had one or more attacks of gall-stone colic, from new attacks, and from the other consequences of biliary calculi. The more frequently the attacks have been repeated, and the more the surfaces, angles, and facettes of the calculi passed induce the belief that there are others remaining in the gall-bladder, the more imperative are the rules for insuring protection. Experience shows that, under the use of the Karlsbad waters, immense quantities of gall-stones are evacuated, with proportionately little difficulty. The same is true of the use of other alkaline mineral waters, of the waters of Vichy, Marienbad, Kissengen, etc. We cannot explain this fact. We do not know whether their efficacy depends solely on the rich formation of a thin, fluid bile, by which the gall-stones are readily washed downward, or whether the bile is rendered so strongly alkaline by the use of these waters as to dissolve the coloring matter and lime, or the cholesterin; but we should not delay prescribing the treatment till its mode of action can be explained. In the treatment of the states induced by gall-stones, *Durande's* remedy also enjoys a great reputation; this consists of ether ʒ iij, and oil of turpentine ʒ ij. According to the original prescription, half a drachm of this is to be given in the morning, and the dose is gradually increased until about a pound of the mixture has been taken. The fact that ether and oil of turpentine dissolve biliary calculi placed in them does not justify the hope that they will dissolve any concrements in the gall-bladder, if they are introduced into the stomach. Hence, if *Durande's* remedy has a favorable influence on the conditions induced by gall-stones, as we must suppose it has, from the recommendations of numerous and good observers, this can only take place in some other way, which is entirely unknown to us. Recently various substitutes for *Durande's* remedy, and variations from the original dose, have been proposed. There is a popular mixture of oil of turpentine, ℈ij, with spirits of ether, ʒ j, which is prescribed, in drop-doses, by *Rademacher* and his followers, not only for gall-stones, but for all possible liver-diseases, whether we know what they are or not.

In the treatment of gall-stone colic, the bold employment of opium deserves the most reliance. We may give twelve drops of laudanum or a quarter of a grain of acetate of morphia at a dose, and repeat it every hour or two till there is slight narcotism. If the patients vomit, so that they cannot retain medicines given by the stomach, we may give subcutaneous injections of a strong solution of morphia, or enemata of laudanum, or let the patient carefully inhale chloroform. Warm baths, also, as well as warm narcotic compresses over the liver, occasionally, appear to moderate the pain and shorten the attack. If this be, nevertheless, protracted, and the region of the liver become very sensitive to pressure, we should apply a number of leeches to the right hypochondrium; in such cases they appear to have a favorable effect, although we cannot understand why. The patient not unfrequently becomes so collapsed that, besides the above-mentioned remedies, we are obliged to give analeptics. Pieces of ice are most efficacious for the severe, and occasionally very obstinate, vomiting. Emetics and laxatives, given during the attacks, increase the pain, and, moreover, they may prove dangerous. On the contrary, after the attack, we should administer mild laxatives for a while, so that any concrements in the intestines may be evacuated as soon as possible.

In inflammations and ulcerations of the bile-passages caused by gall-stones, we must limit ourselves to the treatment of symptoms, as we are unable to remove the exciting cause. Fluctuating abscesses in the abdominal walls should be opened early; any remaining fistulæ should be treated according to the rules of surgery. Obstructions of the intestinal canal by large gall-stones are to be treated as previously advised; severe and distressing pain should be relieved by narcotics.

# *DISEASES OF THE SPLEEN.*

## CHAPTER I.

### HYPERÆMIA OF THE SPLEEN.

ETIOLOGY.—The variations in the amount of blood in an organ may be the more decided, the more yielding its parenchyma and envelope, and the more numerous its vessels, and the thinner their walls. The spleen has a very yielding capsule, its numerous vessels have very thin walls, and appear to communicate with large cavities within. This explains how the spleen may be enormously distended by injections of water, or by blowing up, as well as the fact that, during life, the amount of blood in it may be very much increased, and it may, consequently, be very decidedly enlarged.

The slighter the elasticity of the envelope, and of the walls of the vessels of an organ, the slower the disappearance of distention induced by any temporary cause. If we imagine an organ where the envelope and the walls of the vessels have no elasticity, it would remain permanently enlarged, if once distended by a momentary increase of the blood flowing in, or by a momentary obstruction to that flowing out; just as a wax-tube, that has a fluid passing through it, remains permanently dilated if we momentarily increase the pressure on its inner wall. As the capsule, trabeculæ, and walls of the splenic vessels offer but little opposition to its enlargement, so also, from their slight elasticity, they can only slowly remove any enlargement of the organ. If the spleen be swollen during a paroxysm of intermittent fever, after the subsidence of the paroxysm, it remains enlarged longer than other organs that were enlarged at the same time, but which were richer in elastic elements, and particularly such as had vessels with more elasticity than the vessels and cavities of the liver. We shall hereafter show that the decrease of swelling of the spleen is probably caused by its contractile elements.

In the spleen as in other organs we must distinguish two forms of hyperæmia, *fluxion* and *obstructive engorgement.*

*Fluxion* causes.—1. Splenic enlargements in the *acute infectious diseases;* the enlargement in typhus and intermittent fevers, in the acute exanthemata, puerperal fever, septicæmia, etc. We do not know whether the increased flow of blood into the spleen in these diseases be due to a relaxation of the already yielding tissue of the spleen, or to a paralysis of the muscular elements of the walls of the vessels, and of the trabeculæ. (*Jaschkowitz* observed that, after dividing the branches of the sympathetic going to the spleen, it became very large and excessively vascular. If he only divided some of the nerves, the hyperæmia was limited to the parts of the spleen supplied by the divided nerves.) The manner in which the infected blood may alter the elasticity of the tissue of the spleen, or the contractility of its muscular elements, is just as obscure. The swelling of the spleen in intermittent fever has been explained by saying that, during the chill, the circulation on the surface of the body is decidedly disturbed, and, on account of the ischæmia of the skin, the internal organs, and among these the spleen particularly, are overloaded with blood. But these conditions are of only secondary importance, as is proved by the fact that the amount of splenic enlargement is not at all in proportion to the severity of the chill, that the spleen also enlarges during the hot stage, and finally, because enlargement of the spleen occurs from malarial infection when there is no fever. 2. Fluxion to the spleen occurs in *anomalies of menstruation;* we might repeat of this form all that was said of the occurrence of hyperæmia and hæmorrhage from the gastric mucous membrane arising from the same cause. 3. Injuries, inflammations, and neoplasiæ in the spleen induce fluxions. We may best observe this form of hyperæmia in hæmorrhagic infarction of the spleen (see Chapter IV.).

There is a physiological *engorgement* of the spleen a few hours after every meal, that is, at the time when the lateral pressure in the portal vein is greater from the increased supply of blood coming from the filled intestinal veins, and when the flow of blood from the splenic vein is obstructed. Abnormal congestion is induced by obstruction and closure of the portal vein, such as occur in numerous diseases of the liver, as cirrhosis, pylephlebitis, and others. Since most of these last a long while, besides the hyperæmia of the spleen, we usually find its results which will be spoken of in the next chapter. Engorgement of the spleen is far less constant and excessive in heart and lung diseases, where the flow of blood from the vena cava is obstructed, and this obstruction extends through the vessels of the liver to those of the spleen. It is difficult to explain why, in spite of excessive cyanosis and general dropsy, the spleen often remains of normal size, and not very vascular in heart and lung disease. It is even more remarkable that, in atrophic nutmeg-liver, hyperæmia of the spleen is even absent as a rule.

ANATOMICAL APPEARANCES.—Except in cases where the capsule of the spleen is thickened and unyielding, we find the hypertrophied organ larger and heavier than a healthy spleen. The increase in size and weight may be so great that the organ will have from four to six times its normal weight. The normal spleen of a healthy adult is four to five inches long, three to four inches wide, and one to one and a half inches thick; its weight is about eight ounces. The spleen enlarged by hyperæmia maintains its form, its capsule usually appears tense and smooth, and where the swelling has somewhat subsided, it is occasionally relaxed and wrinkled. The consistence of the spleen is diminished. This is also true of the enlargement of the spleen occurring in malarial diseases, as long as it is recent, and as long as other changes, that will be spoken of hereafter, have not occurred. The enlarged spleen, found in patients who have died of typhus, puerperal fever, septicæmia, etc., is often so soft that, when cut through, the parenchyma flows off like pulp. In judging of the consistence of this tumor, we must, however, remember the early decomposition of the bodies. The color of the spleen is darker in proportion as the hyperæmia is recent and excessive. In the most recent cases and in high grades of hyperæmia, the parenchyma often looks like a blackish red-blood coagulum, later it appears lighter colored, or from admixture of pigment is somewhat gray.

On microscopical examination, we find no foreign elements with the normal cells of the spleen pulp, and numerous blood-corpuscles, so that we have no right to refer this enlargement of the spleen to a process of inflammation and exudation. Acute splenic enlargement appears to depend either solely on increase of the blood contained in it, and serous infiltration of the tissue, or on a coincident temporary increase of the substance of the spleen.

When the hyperæmia has existed a long time, the increase of the pulp of the spleen is unmistakable; it greatly changes the appearance and consistence of the organ; the spleen remains permanently enlarged, and we have the state called "chronic spleen tumor" or hypertrophy of the spleen, which we shall describe in the next chapter.

SYMPTOMS AND COURSE.—Hyperæmic swelling of the spleen almost always develops without the patient complaining of pain; usually he is only sensitive on deep pressure in the left hypochondrium. This observation corresponds to the general experience that tension of tissues which are very expansible causes little pain, while tension of membranes, ligaments, etc., which are stretched with difficulty, excites severe pain. If, during an intermittent, a typhus or similar state, the patients of their own accord complain of pain in the region of the spleen, it may either be because the capsule of the spleen has become thickened and unyielding from former disease, or from the occurrence in the spleen or its capsule of inflammatory processes, which may also occur during these affections.

In most cases there are no other subjective symptoms either, at least such as can be certainly referred to hyperæmia of the spleen, and not to the original disease. Hence the splenic affection would almost always be overlooked if the physician did not know that it very constantly occurred in certain diseases, and if he did not examine each case by palpation and percussion, to find whether any enlargement of the organ existed. I will call attention to one symptom of excessive hyperæmia of the spleen, which I think can be readily explained, and referred to mechanical causes. Experience shows that some patients with intermittent fever are very pale and anæmic after a very few paroxysms, and that the pallidity of the skin and mucous membranes disappears in a few days when a few doses of quinine have arrested the paroxysms. This symptom cannot depend on the rapid consumption of the blood and its speedy restoration. Although the temperature rises very high during the paroxysm of the intermittent, and we know that high fever is accompanied by decided and rapid consumption of the blood, still in no other disease where the temperature reaches the same height, and remains there even longer than in intermittent, does the patient become anæmic in so short a time. On the other hand, if a continued and high fever has caused poverty of the blood, the symptoms disappear far more slowly than does the paleness resulting from a few paroxysms of intermittent fever. My observations and those of others, particularly of *Griesinger*, show that the rapidity with which the symptoms of anæmia develop and the grade that they attain are in direct proportion to the rapidity with which the spleen enlarges, and to the grade that this enlargement reaches; that, particularly in children, in whom the spleen usually acquires a proportionally large size after very few paroxysms, threatening symptoms of excessive hyperæmia develop early, but disappear very quickly after the arrest of the paroxysms and the subsidence of the tumor. Hence it can hardly be doubted that the appearance and disappearance of these anæmic symptoms are associated with the occurrence and subsidence of the hyperæmia of the spleen. It is not probable that the excessive anæmia, which develops in a few days in intermittent fever, depends on disturbance of the spleen, caused by the hyperæmia, although, in severe disease of the spleen, the blood is gradually impoverished, apparently from disturbance of the influence of that organ on the formation of the blood. On the other hand, although not positively certain, it is very probable, from what was said above, that the overloading of the spleen with blood induces anæmia in the rest of the body, and consequently that the pallor of the patient depends less on impairment of the quality of the blood than on its abnormal distribution. We may compare the effect that overloading of the spleen with blood has on the rest of the body, to that induced by a large aneurism filled with blood, or by the overloading of one of the lower extremities with blood from the application of *Junod's* boot. If, after the cessation of the intermittent paroxysms, or after the administration of quinine, the spleen regains its normal size from its elasticity, or from the contraction of the irritable elements of the tissue, the anomalous distribution of the blood will cease. This explains how the redness of the skin and lips, which has been lost during the intermittent fever, may return in a few days.

The hyperæmia of the spleen that occurs in the course of typhus and similar diseases usually subsides when they have run their course, without leaving any structural change. The case is different where the enlargement depends on intermittent or other fluxionary or obstructive hyperæmia, if it continues for a long time, on account of continued action of the exciting cause. In the next chapter we shall attempt to prove that so-called hypertrophy of the spleen is a necessary result of long-continued hyperæmia. In very rare cases hyperæmia of the spleen proves fatal, from rupture of the distended organ. This termination has been seen both in paroxysms of intermittent fever, and in typhus and cholera. Death results with the symptoms of internal hæmorrhage either immediately after the rupture of the spleen, or else not for several hours or days.

Physical examination furnishes the most important, and often the sole means, of diagnosis of hyperæmia of the spleen. While speaking here of the physical signs that occur in hyperæmic swelling of the spleen, we shall say a few words concerning the physical diagnosis of diseases of the spleen in general.

The upper part of the spleen lies in the hollow of the diaphragm, and is covered by the lower border of the left lung; its lower part lies in immediate apposition with the wall of the thorax, and normally does not quite reach to the angle of the ribs. Percussion is frequently the sole means of recognizing an enlargement of the spleen, as that organ frequently does not reach below the margin of the ribs even when decidedly enlarged. The normal dulness of the spleen extends from the upper margin of the eleventh rib to the ninth rib; anteriorly it is bounded by a line drawn from the anterior end of the eleventh rib to the nipple; posteriorly the spleen-dulness cannot be defined from that of the left kidney. Its greatest thickness is about two inches. If the spleen enlarges, its dulness partly extends forward and downward, and partly, by pushing the diaphragm before it, upward, but rarely higher than the fifth rib. If the intestines be distended by gas, and the abdominal walls be tense, the dulness extends more upward; if the intestines be empty, and the abdominal walls relaxed, it extends more anteriorly and downward. The spleen-dulness changes its place during respiration, being about an inch lower on deep inspiration, and about an inch higher on full expiration. When the patient lies on the right side, the spleen-dulness is less; hence it is well to examine the patient in different positions, and, if we wish to determine whether the extent of dulness varies from time to time, we should carefully note in what position of the patient the previous examination was made.

If tumors of the spleen project below the ribs, and be not too soft, we may readily recognize them, and distinguish them from other tumors by *palpation*. While they have only a moderate extent, they can often only be felt when the patient inspires deeply; on expiration, they disappear beneath the ribs. When the growth is considerable, the swelling gradually extends from the left hypochondrium, obliquely, toward the navel. It almost always maintains the characteristic form of the spleen, particularly the shallow excavation in the anterior rounded edge. The tumor follows the movements of the diaphragm, can be readily moved, and changes its position with the position of the body. When the spleen is very large, instead of taking an oblique direction, it often passes directly downward into the pelvis, and, becoming less movable, no longer follows the motions of the diaphragm. The spleen-dulness may disappear from the thorax, as a result of elongation of the ligament attaching the spleen to the diaphragm, from the weight and size of the enlarged organ.

In some cases even *inspection* shows the enlargement of the spleen by a prominence of the left hypochondrium and left half of the abdomen, in which the contours of the spleen are occasionally noticed.

The spleen enlarged by hyperæmia does not by any means always extend below the ribs, and, even when it does, may escape detection by palpation if it be very soft.

If hyperæmia of the spleen accompany abdominal typhus, on percussion we usually find the dulness extending more backward, on account of the meteorism of the intestines; if, on the other hand, it accompany an intermittent, the dulness rather extends toward the axilla, and occupies the left hypochondrium.

TREATMENT.—In accordance with what we have said of its course, hyperæmia of the spleen is rarely the object of treatment. If we can remove the original disease, it almost always disappears in a short time without our interference. For those forms that do not subside spontaneously, particularly for those caused by malaria, we have a very effective remedy. If we avoid all exaggeration, and rely on trustworthy observations, it still appears probable that, in the whole materia medica, there are very few remedies that act certainly against any diseases as quinine and the preparations of Peruvian bark do in the hyperæmia of the spleen resulting from malaria. We do not know whether quinine acts by directly inducing contraction of the muscular elements of the spleen, or whether it acts as an antidote to the malaria, and, by destroying the cause, arrests its results, or whether it removes the hyperæmia of the spleen in some other way; but we do know that, if, after the attacks have ceased, the spleen remains enlarged or decreases in size very slowly, the disappearance of the swelling is caused or hastened by giving large doses of quinine. It is certain that the effect of quinine on hyperæmia of the spleen cannot be solely due to its arresting the fever.

According to *Fleury's* observations, the enlarged spleen is reduced several centimetres during the application of the cold douche. *Fleury* refers to the observations of *Andral* and *Piorry* in support of his assertions. This procedure deserves attention in treating hyperæmia of the spleen, if circumstances permit it.

## CHAPTER II.

### HYPERTROPHY OF THE SPLEEN—CHRONIC ENLARGEMENT OF THE SPLEEN—ANÆMIA ET CACHEXIA SPLENICA.

ETIOLOGY.—I consider the name of hypertrophy of the spleen as best suited for that form of enlargement of the organ, where its size and weight are increased, without any change in its texture being observable. But in the form of enlargement of the spleen under consideration, the increase of the trabecular tissue is unimportant, as compared with the far greater increase of the pulp, and the latter forms the most important factor in the enlargement.

With our present knowledge, in most cases we cannot fully answer the question as to whether the increase of the pulp of the spleen be due to excessive formation, a "hyperplasia" of its cellular elements, or to their abnormal accumulation as a result of obstruction to their escape. It is almost universally believed that the spaces between the trabeculæ of the spleen, which contain the pulp, in some way communicate with the vessels. If this view is the true one, that is, if the blood flows through those spaces, and constantly carries cellular elements out of them, just as the lymph flowing through the cells in the lymphatic glands carries cellular elements from them into the lymph, it is most probable that, if the stream of blood in the spleen is much retarded, the pulp of the spleen must increase, because less of its cells are carried into the blood. The increased size of the current due to distention of the vessels, and still more to distention of the intertrabecular spaces from the hyperæmic swelling, very decidedly retards the current of blood in the spleen; and, since long-continued hyperæmia of the organ constantly induces hypertrophy, it is very probable that this form of hypertrophy of the spleen, at least, is due to accumulation of the pulp, and not to its excessive formation.

When speaking of leuchæmia (see appendix to this chapter), we shall mention a degeneration of the spleen which cannot be anatomically distinguished from the one under consideration, but which must be separately considered, because the changes it induces in the composition of the blood are so peculiar that we must suspect a decided functional difference. We shall show that in leuchæmia the enlargement of the spleen is not due to retention of the cellular elements, but to their multiplication.

Among the different forms of fluxionary hyperæmia, those caused by malaria most frequently induce hypertrophy of the spleen; and we find chronic enlargement of that organ, not only when the malarial infection assumes the form of an intermittent fever, but also when it induces remittent fever, or a chronic disease, without paroxysms. Where malaria is endemic, numbers of persons have immense spleens, and it even appears as if the largest tumors are found in persons who escape regularly recurring paroxysms of fever.

Among the congestive hyperæmias, those caused by cirrhosis of the liver and obliteration of the portal vein most frequently result in hypertrophy of the spleen, and this is the strongest proof of the correctness of the hypothesis that this form of spleen-disease is caused in a purely mechanical way, by accumulation of cellular elements, as a result of obstructed escape of the blood.

Recently a number of cases of decided hypertrophy of the spleen, occurring without any perceptible cause, have been observed and described. As the anatomical appearances of these "idiopathic" enlargements of the spleen appeared to correspond entirely with those of leuchæmic spleens, of which we shall hereafter speak, and as the symptoms observed during life (except the increase of the colorless corpuscles of the blood) were very similar to those of leuchæmia, the name of pseudo-leuchæmia has been proposed for this disease, while other observers designate it as anæmia or cachexia splenica. In the so-called pseudo-leuchæmia, besides the affection of the spleen, there is almost always an analogous affection of the lymphatic glands, that is, their decided enlargement depending on simple increase of the normal elements. In some cases the spleen is most affected (splenic form), in others the lymphatic glands (lymphatic forms).

ANATOMICAL APPEARANCES.—As a result of hypertrophy, the spleen may become so enormous as to measure one foot to a foot and a half long, over six inches wide, and over four thick. It may attain a weight of twelve pounds or more. The form of the enlarged organ is not changed; its resistance is increased, so that it occasionally becomes as hard as a board. In recent cases, the color of the parenchyma is dark-brownish red, in older ones it is usually the color of the muscles or pale red. If the hypertrophy has resulted from malarial affection, the pale, homogeneous, and dry cut surface usually has a gray tinge, or we find dark spots in it. In the appendix to this section, when speaking of melanæmia, we shall more particularly describe the extensive deposits of pigment which occasionally remain in the spleen after pernicious intermittent fever. The capsule of the hypertrophied spleen is usually thickened, cloudy, and not unfrequently adherent to its surroundings. The thickened and rigid trabeculæ of the spleen appear as white striæ on the cut surface.

Besides the closely-packed normal elements of the pulp of the spleen and interspersed pigment, microscopical examination does not show any foreign formations. In the idiopathic as well as in the leuchæmic enlargements of the spleen, we not unfrequently find wedge-shaped masses similar to the hæmorrhagic infarctions to be described in Chapter IV.

SYMPTOMS AND COURSE.—While examining a previously healthy and fresh-looking person for an acute disease, we not unfrequently find a great enlargement of the spleen. Such cases prove either that a morbidly-enlarged spleen can fulfil its functions, or that other organs may act vicariously for it. In favor of the latter supposition is the well-known fact that dogs, from which the spleen has been removed, may live a long while, be well nourished, propagate the species, etc.

The relative or even perfect good health, often found in persons with old enlargement of the spleen, is very analogous to the perfect health and blooming looks of persons who, for years, have had enlarged lymphatic glands in the neck or elsewhere. If we carefully examine the history we shall find that when the enlargement of the spleen or the swelling of the glands occurred, the patient's state was not by any means so fair as at the time of examination, but that there were at that time more or less decided signs of anæmia and cachexia. This appearance of anæmic symptoms, at the time of the occurrence of the enlargement of the spleen and lymphatic glands in question, and the subsequent disappearance of the anæmia, in spite of the continuance of the tumors, are perfectly in accordance with the doctrine that the spleen and lymphatic glands are the places where the blood-corpuscles are formed. Apparently the state of affairs is as follows: When the cells formed in the intertrabecular spaces of the spleen, or in the cells of the lymphatic glands, are retained in any way, and are not normally borne along by the blood and lymph, they accumulate in these places; the spleen or the lymphatic glands, as the case may be, swell up and the blood becomes poor, as the used-up blood-corpuscles are not replaced by others. If the obstruction to the passage of the young cells into the circulation be removed, the growth of the spleen and lymphatic glands ceases, and the blood is gradually improved by a sufficient supply of young cells, even if the enlarged spleen or lymphatic glands do not decrease in size.

If the spleen continues to enlarge for a long time, there is great impoverishment of the blood. The patients become excessively dull and feeble; their skin grows waxy, and brunettes acquire a clayey look ("splenetic"). The lips and visible mucous membranes also appear very pale and bloodless. Since the number of blood-corpuscles aërated in the lungs is diminished, the usual number of inspirations no longer suffices to supply the blood with oxygen, and to remove the carbonic acid. Hence the patients are short-breathed, and, where bodily exertion and similar causes increase the demands for oxygen, they suffer from dyspnœa. The nutrition of the walls of the capillaries also suffers from the impoverishment of the blood, and they become morbidly fragile, causing a hæmorrhagic diathesis. Capillary hæmorrhages occur without perceptible cause, apparently spontaneously, particularly epistaxis, and we have petechiæ from hæmorrhages in the tissue of the skin. The common assertion, that in disease of the spleen the bleeding usually proceeds from the left nostril, is false. If the disease continues to progress, the anæmia, or more properly the hydræmia, finally increases to a so-called dropsical crasis; there is usually œdema of the lower extremities, and in severe cases there is gneeral dropsy.

If the above symptoms be observed in a patient who has enlargement of the spleen, as a result of malarial infection, or as a complication of cirrhosis of the liver, it may be difficult to decide what part of the symptoms is due to the disease of the spleen, and what to the original disease; nevertheless, the fact that the grade of the hydræmia, both in the malarial infection and in the cirrhosis, has a certain relation to the amount of enlargement of the spleen, justifies us in not rating too low the effect of the spleen-disease on the impoverishment of the blood even in such cases. This influence is far more striking in cases where he enlargement of the spleen is independent of other diseases, and ocurs as a primary and idiopathic affection.

I would find it difficult to make a distinction between the so-called pseudoleuchæmia (a very unsuitable name) and the chronic spleen-disease which has been recognized for ages and has frequently been carefully described. Steadily increasing impoverishment of the blood, great paleness of the skin and visible mucous membranes, hæmorrhagic diathesis, and in severe cases dropsical symptoms, together with the enlargement of the spleen which is often decided, form the symptoms of the pretended new disease. These symptoms, which are often considered as pathognomonic of spleen-disease, reach a very high grade, and finally cause death in idiopathic enlargement of that organ: first, because we cannot arrest the enlargement; secondly, because in many cases the affection of the spleen is accompanied by a similar disease of the lymphatic glands. In most of seven cases of idiopathic enlargement of the spleen that have come under my own observation, and have been reported by my assistant, Dr. *Müller*, in the "Berliner klinischen Wochenschrift," besides the enlargement of the spleen, there was decided swelling of numerous lymphatic glands. After what we have previously said, it cannot appear strange that the simultaneous disease of the spleen and of the lymphatic glands, which also participate in the formation of the blood-corpuscles, is a very dangerous complication, and that it should induce the highest grade of impoverishment of the blood.

As hypertrophy most frequently causes very great enlargement of the spleen, the enlargement may be recognized, on physical examination, both by inspection and palpation as well as by percussion. The tumor preserves the characteristic form of the spleen; its resistance is increased, although not so much so as lardaceous spleen, which we shall describe in the next chapter.

TREATMENT.—Recent hypertrophy of the spleen, resulting from malarial infection, calls for the same treatment that was recommended in chronic hyperæmia of that organ. Change of residence and the use of the preparations of Peruvian bark, particularly of quinine, are exceedingly useful; but the patient should not return to the malarial region too soon, and should perseveringly use the quinine for a long time. Even in old cases of hypertrophy we should try the effect of these remedies. Of the numerous derivatives recommended for the treatment of chronic enlargement of the spleen, the cold douche alone appears serviceable, while blistering-plaster, issues, the actual cautery, etc., over the spleen, promise but little benefit. The preparations of iron are very extensively and very properly used in the treatment of this affection; among these, muriate and iodide of iron are particularly celebrated. We will not undertake to say whether they have any influence in decreasing the size of the spleen, or whether their beneficial effect is due to the improvement of the quality of the blood. The effect is best when we combine the preparations of iron with quinine, or if we order the use of chalybeate mineral waters in some mountainous region, and at the same time let the patient take quinine continually. Treatment does no good in hypertrophy of the spleen due to cirrhosis, pylephlebitis, etc.

## CHAPTER III.

### LARDACEOUS SPLEEN—AMYLOID DEGENERATION OF THE SPLEEN.

ETIOLOGY.—In lardaceous spleen the walls of the vessels and the cellular elements of the pulp degenerate just as the liver-cells do in lardaceous liver. More rarely, in the so-called sago-spleen, the pulp is unaffected, the cells and nuclei only undergoing the lardaceous or amyloid degeneration.

Regarding the etiology of lardaceous spleen, we may refer to what was said of the analogous disease of the liver. The dyscrasiæ there mentioned, scrofula, rachitis, tertiary syphilis, and mercurialism, also induce amyloid degeneration of the spleen. Lardaceous spleen only exceptionally complicates tuberculosis; on the other hand, it occurs quite frequently in malarial diseases, although far more rarely than simple hypertrophy.

ANATOMICAL APPEARANCES.—Amyloid degeneration may cause as great enlargement of the spleen as results from the hypertrophy mentioned in the last chapter. The lardaceous spleen is very heavy, and excessively hard; if we attempt to bend it, we find that, besides being hard, it is very friable. The color is usually a pale violet red; the little blood contained in it is watery; the cut surface is homogeneous, smooth, dry, and has a lardaceous, waxy lustre. On microscopical examination, we find the cellular elements of the pulp enlarged, of a dull color, with pale, homogeneous contents. On adding a solution of iodine, the preparation becomes yellowish red, and on a further addition of sulphuric acid it becomes violet and blue.

If the degeneration be limited to the *Malpighian* bodies, the organ is not usually so much enlarged. On incising the spleen, we find roundish, gelatinous granulations, resembling swollen sago, scattered through the moderately firm parenchyma. Microscopical examination shows that the cells and nuclei of the *Malpighian* bodies are changed in the manner above described for the spleen-pulp.

SYMPTOMS AND COURSE.—In lardaceous degeneration of the spleen also, the patients are very anæmic and cachectic. Epistaxis, petechiæ, and dropsy, are more frequent than in simple hypertrophy. And in this form of enlargement of the spleen it is even more difficult than in the preceding ones, to say how far these symptoms depend on the original disease, and how far on the degeneration of the spleen. Moreover, besides the lardaceous degeneration of the spleen, there is usually an analogous affection of the liver and kidneys, which increases the impoverishment of the blood. Physical examination often shows enormous enlargement of the organ.

In spite of the similarity of the symptoms, the diagnosis between the two forms of chronic enlargement of the spleen is usually easy. The occurrence of enlargement of the spleen in the course of one of the above-mentioned diseases, coincident disease of the liver and kidneys, steady growth of the tumor, which never recedes, and its uncommon firmness, speak in favor of lardaceous spleen, and against simple hypertrophy.

**Treatment.**—Treatment is useless in lardaceous spleen. It is true, iodide of iron has a certain reputation, and it is possible that the impoverishment of the blood, and the dyscrasia causing the splenic disease, may be improved by its use; but, even if this happens, it is not probable that the size of the spleen will decrease, or that its structure will again become normal.

## CHAPTER IV.

### HÆMORRHAGIC INFARCTION AND INFLAMMATION OF THE SPLEEN—SPLENITIS.

**Etiology.**—In no organ is hæmorrhagic infarction more frequent than in the spleen, and in most cases it undoubtedly proceeds from obstruction of a small artery by an embolus. The size of the splenic artery and the rapidity with which the blood flows through it (a necessary result of the slight obstruction the blood meets in the spleen) explain why emboli from the aorta most readily enter the splenic artery. The emboli usually originate in the left heart, and are fibrinous coagula that have been deposited on rough places on the valves, in endocarditis and valvular disease, and have subsequently been washed off by the blood. When autopsy reveals extensive valvular disease, with roughness or rupture of the valves and chordæ tendineæ, it is almost a rarity not to find old or recent infarctions in the spleen. Far more rarely the emboli come from necrosed spots in the lungs, and have passed through the pulmonary vein and left heart, before entering the aorta and splenic artery.

Hæmorrhagic infarctions of the spleen also exceptionally occur in those diseases which usually induce only excessive hyperæmia of that organ. We find it as well in malarial infection as in typhus, septicæmia, and the acute exanthemata. *Jaschkowitz* found that, when he had divided some of the nerves of the spleen, the pathological changes usually called hæmorrhagic infarctions sometimes occurred in the parts of the spleen supplied by the nerves that had been divided.

As it is doubtful whether the parenchyma of the spleen be normally separated from the current of blood by the walls of the vessels, it becomes a question whether hæmorrhagic infarction be due to an escape of blood from the vessels, or whether it be not rather owing to a coagulation of the blood in the vessels and in the intertrabecular spaces. In the latter case hæmorrhagic infarction would represent thrombus of those spaces, as it were, and, like other thrombi, would be the result of a retardation of the current of blood.

Primary *inflammation of the spleen* is an exceedingly rare disease. Even injuries are more apt to cause rupture than inflammation of that organ. Consecutive inflammation and suppuration of the spleen are more frequently induced by hæmorrhagic infarctions, particularly by those occurring during infectious diseases. If the infarction be a primary coagulation in the vessels and in the intertrabecular spaces (which is at least as probable as the opposite view), then the splenitis would hold the same relation to the infarction that phlebitis does to thrombus of the veins.

ANATOMICAL APPEARANCES.—Hæmorrhagic infarctions of the spleen are roundish, or more frequently wedge-shaped collections (with the bases outwardly) of the size of a pea or a hen's egg. At first they are dark brown, or brownish red, and quite hard. The entire spleen is enlarged by fluxionary hyperæmia, the peritonæum over the infarction is freshly inflamed. Later the collections become of a dirty-yellow color, starting from the centre. The final result varies: either fatty degeneration occurs, and the mass is reabsorbed, and in place of the infarction we have a retracted, callous cicatrix, or else a yellow, cheesy mass, which may become calcareous, remains; or, lastly, the infarction softens, and there is an abscess filled with detritus, in which pus-corpuscles also appear after a time. The latter course chiefly is taken by those cases where small but numerous infarctions occur in typhus and similar diseases.

We know nothing about the anatomical changes in primary *splenitis* before it has induced abscess. Occasionally we find the abscess incapsulated in a proliferation of connective tissue; in other cases it is surrounded by disintegrated connective tissue, or the entire spleen, except its capsule, has become disintegrated, so that the latter forms a large sac, which is filled with pus. Finally the capsule of the spleen is perforated, and the contents of the abscess either enter the abdomen, or, if the capsule has previously become adherent to the parts around, it enters some neighboring organ. Cases have been reported where the pus, from an abscess of the spleen, has entered the stomach, or colon, or, passing through the diaphragm, has reached the pleural sac, or has perforated outwardly, through the abdominal walls. It is only in very rare cases that an abscess of the spleen dries up, its contents becoming inspissated and calcareous, or breaking through the capsule, and being evacuated.

SYMPTOMS AND COURSE.—Where hæmorrhagic infarction occurs in the course of an infectious disease, it is almost always first recognized at the autopsy. On the other hand, where it accompanies heart-disease it can often be recognized during life. If endocarditis or valvular disease has been diagnosed in a patient, and he complains of pain in the left hypochondrium, which is increased by pressure; if there be vomiting also, and physical examination shows enlargement of the spleen, which did not exist a few days previously, we may decide that there is hæmorrhagic infarction of that organ. The pain is due to the partial peritonitis which almost always accompanies the infarction. The vomiting is a sympathetic symptom. And, lastly, the enlargement of the spleen is the result of fluxionary hyperæmia. In almost all the cases I have seen, the above combination of symptoms commenced with a chill, and was accompanied by repeated chills. We have already said that these do not justify us in deciding on a septicæmic affection.

Most cases of abscess of the spleen that have been described have been latent, and were not recognized during life. Chills, hectic fever, cachectic appearance, rapid emaciation, and dropsical symptoms showed that there was some severe disease, but its nature was not discovered. If, besides these symptoms, there was pain in the left hypochondrium, and enlargement of the spleen could be detected, it was occasionally possible to form a probable diagnosis. Distinct fluctuation was very rarely found.

If the abscess perforate the capsule of the spleen, and its contents be emptied into the abdomen, we have the symptoms of diffuse peritonitis; or, if they have entered a capsulated space, those of a circumscribed peritonitis. If its contents enter the stomach or colon, mixed blood and pus are vomited or passed at stool. If the perforation take place into the pleura, into the lungs, or outwardly, the symptoms are similar to those described for perforation of abscesses of the liver in these directions.

TREATMENT.—In hæmorrhagic infarction, as in suppurative splenitis, treatment is of no avail. We can only give palliatives for the most urgent symptoms. Where the pain is severe, we should order local abstraction of blood and cataplasms; for the sympathetic vomiting carbonates and bicarbonates of the alkalies, or, if it be very obstinate, we may give narcotics; fluctuating abscesses should be opened early, and with the same precautions as in abscesses of the liver.

## CHAPTER V.

### TUBERCULOSIS, CARCINOMA, HYDATIDS OF THE SPLEEN.

TUBERCULOSIS of the spleen occurs, sometimes, under the form of numerous gray miliary tubercles, as one part of miliary tuberculosis; sometimes it complicates tuberculosis of the intestines and mesenteric glands, under the form of yellow, cheesy conglomerations of tubercle, which rarely attain the size of a hazel-nut, and only exceptionally break down and form vomicæ. Tuberculosis of the spleen cannot be recognized during life, and hence there can be no question about its treatment.

*Carcinoma* also is rarely observed in the spleen. Medullary carcinoma is the only one of the various forms that ever occurs here. In almost all the cases on record, the disease did not affect the spleen primarily, but accompanied carcinoma of the stomach, liver, or retroperitoneal glands. The spleen may acquire an uneven, nodular appearance from large cancerous tumors. From the great rarity of carcinoma of the spleen, it should be the last disease thought of when we are trying to determine the nature of an enlargement of that organ; we should only make the diagnosis of carcinoma of the spleen when the enlargement no longer retains the characteristic form of the spleen, but has an irregular, nodulated surface, and when there is at the same time carcinoma of the stomach or liver.

*Hydatids* of various size and number are also seen rarely in the spleen, and almost exclusively in cases where they also occur in the liver. During life they can only be recognized when hemispherical protuberances, with the previously-described peculiarities of hydatid cysts, can be felt on the enlarged spleen.

# APPENDIX TO THE DISEASES OF THE SPLEEN.

WE do not propose to treat of leuchæmia and melanæmia, in the second volume, among diseases of the blood, but to class them among diseases of the spleen, as they generally depend on affections of that organ. But, as there are also cases of leuchæmia, and even some of melanæmia, where the blood-affection cannot be referred to disease of the spleen, these affections must be described in an appendix.

## CHAPTER I.

### LEUCHÆMIA (LEUCOCYTHÆMIA—*Bennett*).

ETIOLOGY.—A temporary increase of the colorless corpuscles of the blood takes place in a number of physiological and pathological conditions, as, during pregnancy, inflammatory diseases, or after great loss of blood. This variation of the blood, from its normal state, is no more an independent disease than hyperinosis and hypnosis, anæmia, or hydræmia, but it is the result of various states.

The case is different with leuchæmia. This very interesting disease is defined by *Virchow* as a "change in the constitution of the tissue of the blood," the blood being classed among the tissues; in it white corpuscles are to a great extent formed instead of red ones, so that the number of the former increases, while that of the latter diminishes. We found our description on the classical work of *Virchow*, who has shown that leuchæmia may depend either on disease of the spleen, or of the lymphatic glands, and that there are two forms of leuchæmia, the *splenic* and *lymphatic*. The changes of the spleen in the former, and of the lymphatic glands in the latter, consist chiefly in an increase of the cellular elements composing the pulp of the spleen, or filling the cells of the lymphatic glands.

Since in leuchæmia we find the blood loaded with the elements, whose accumulation in the spleen and lymphatic glands causes the swelling of these organs, it would appear that the leuchæmic tumors are due to an increased formation of cellular elements, and not to their retention, as we thought was probable in the other forms of enlarged spleen and lymph glands. It is a question whether the cells, so plentifully formed in the spleen and lymphatic glands during leuchæmia, differ in any way from those formed under normal circumstances. If we could suppose that only the white corpuscles of the blood originated from the colorless cells of the lymph and spleen pulp, leuchæmia might be regarded as a simple hyperplasia. Although the transformation has not been directly observed, still it cannot be doubted that, under normal circumstances, the red corpuscles also originate from the colorless lymph-corpuscles, and form colorless cells of the spleen-pulp; hence, in explaining leuchæmia, where this transformation is much limited, we must suppose that the numerously-formed cells do not possess the power of becoming red blood-corpuscles.

In certain cases of this disease, described by *Virchow*, *Friedreich*, and *Böttcher*, other organs also, as the liver, kidneys, intestinal mucous membrane, and pleura, produced lymphatic elements at circumscribed spots; so that, as *Virchow* says, in these cases there was not only a lymphatic dyscrasia, but at the same time a lymphatic diathesis.

The etiology of leuchæmia is entirely obscure. The disease is met with in both sexes, but more frequently in males than in females; it is very rare in childhood. Most cases recorded have affected persons of middle age. No connection has been proved between this disease and malarial infection, or scrofula. In a few cases the disease appeared to have a certain relation to menstruation and to the puerperal state.

ANATOMICAL APPEARANCES.—While, in normal blood, there are about three hundred and fifty red blood-corpuscles to one white one, in leuchæmia the number of white corpuscles may become so much increased, and that of the red ones so much diminished, that the former will become a sixth or even half as many as the latter. In the *splenic* form of the disease the white blood-corpuscles are not distinguishable from those of normal blood; they are distinct, well-developed cells. In the *lymphatic* form, on the other hand, *Virchow* and other observers found numerous free nuclei and small cells, both of which corresponded exactly with the elements found in the lymphatic glands. If the spleen and lymphatic glands were diseased at the same time, if the spleen-disease prevailed, there were more of the larger, cellular elements in the blood; on the other hand, the more extensive the disease of the glands, the more numerous were the small lymphatic elements. Examinations of leuchæmic blood have shown that its specific gravity is much less than that of normal blood: while the latter may be considered as 1055, that of leuchæmic blood was found to be from 1036 to 1049. The diminution of the specific gravity of the serum of the blood was slighter and less constant. In the leuchæmic blood the proportion of water had increased, while, in spite of the increase of the white corpuscles, that of the solid constituents was decreased, as a result of the excessive diminution of the red corpuscles. This, together with the low specific gravity of the white blood-corpuscles, explains the diminution of the specific gravity of the blood as a whole. The albumen, fibrin, and salts of the blood show no decided or constant anomaly. The diminution in the amount of iron, which is considerable, is explained by the deficiency in red blood-corpuscles. And lastly, in leuchæmic blood, *Scherer* found certain constituents of the splenic fluid, such as hypoxanthin, lactic, formic, and acetic acids, and a body whose reactions corresponded with those of glutin; however, other constituents that *Scherer* has found in the spleen-fluid, particularly uric acid, leucin, etc., have not been found in leuchæmic blood.

On autopsy of persons who have died of leuchæmia in the heart, particularly the right one, and in the large blood-vessels, we often find yellow or yellowish-green, soft, smeary coagula, like thickened pus. In the smaller branches of the pulmonary artery, also, and in the veins of the heart and cerebral membranes, discolored puruloid contents have occasionally been found. The proportion of white blood-corpuscles varies in blood taken from different parts of the body. In that from the right heart, vena cava, and pulmonary artery, it is greater than in that from the left heart, and, in a case observed by *De Pury*, it was twice as great in the splenic as in the jugular vein.

In most of the cases of leuchæmia that have been published, the spleen was found greatly enlarged; its weight not unfrequently reached five to seven pounds, or more. In some cases the resistance of the enlarged spleen was increased but little, or not at all; in others (apparently older cases) it was decidedly greater. There was always plenty of spleen-pulp present; the thickened trabeculæ formed white striæ through it. Microscopic examination showed "the normal elements, only they were more closely packed together" (*Virchow*), just as in the above-described hypertrophic enlargement of the spleen, with which the leuchæmic also agrees in its general appearance. In most cases the capsule of the spleen was thickened, and was often adherent to the parts around. In many cases, besides the hypertrophy, there were recent or old hæmorrhagic infarctions in the spleen.

In the lymphatic form, the lymph-glands often formed immense tumors. Of the glands situated within the body, chiefly the mesenteric, lumbar, and epigastric have been found enlarged; of the peripheral, the cervical, axillary, and inguinal glands. Usually, the spleen was also diseased, but *Virchow* saw one case where the spleen was of normal size. I myself have seen a case on which there was no autopsy, but in which no decided enlargement of the spleen could be observed during life, while the lymphatic glands were enormously enlarged. In all the cases the enlarged lymphatic glands were quite soft and pale, their surface smooth and watery-looking, the cortical substance was particularly swollen, in some cases to the thickness of one-half to three-fourths of an inch; it had a homogeneous, almost medullary, appearance, and, on pressure, evacuated a turbid, watery fluid. Microscopic examination showed that the enlargement was entirely due to an excessive formation of cells, nuclei, and granules, similar to those occurring in normal glands. In most cases of this disease, the liver was found enlarged; it was occasionally soft, but usually hard and dense.

An exceedingly interesting pathological new formation of lymphatic elements, outside of the lymph-glands, has been observed in some cases of leuchæmia. In two cases, in the parenchyma of the liver, and in one case in the kidneys also, *Virchow* found small white spots, from which, on pressure, there was evacuated a whitish fluid, consisting only of closely-packed free nuclei, and some small cells, which were almost filled by their nuclei. The new formation was enclosed by a fine membrane, could be quite readily freed from the surrounding parenchyma, and appeared to come from the walls of the blood-vessels and bile-ducts. *Böttcher* observed a similar case. And, in one case of leuchæmia, *Friedreich* found extensive proliferation of nuclei and small cells, not only in the liver and kidneys, but also at circumscribed spots in the pleura, and in the gastric and intestinal mucous membrane, which caused partial thickenings of the pleura, and numerous elevations, of varied extent and prominence, in the stomach, small intestines, and rectum. *Friedreich* also succeeded in proving that the leuchæmic tumors of the pleura and intestinal mucous membrane originated from the connective-tissue corpuscles of those membranes.

SYMPTOMS AND COURSE.—Usually the first symptoms of leuchæmia are swelling of the abdomen, a feeling of pressure and fulness in the left hypochondrium, and other signs of enlargement of the spleen. The enlargement has either come on without pain or fever, so that the time of its occurrence could not be dated, or it has taken place at intervals, during which there was pain in the region of the spleen, and the patient was feverish. And in the lymphatic form, also, the enlargement of the glands in the neck, axilla, etc., which has taken place slowly, or at intervals, first calls attention to the disease. In a few well-observed cases, which throw a very clear light on the dependence of the dyscrasia on the disease of the spleen and lymphatic glands, it was found that the enlargement of the spleen and glands existed for months and years before the disorder of the blood showed itself.

As the blood becomes impoverished in red corpuscles, the patient becomes pale and cachectic; and as the corpuscles not unfrequently become fewer in leuchæmia than in the highest grades of chlorosis, the patients have a waxy appearance in typical cases. There are also almost always complaints of want of breath and hastened respiration, for which symptoms no sufficient explanation can be found in the respiratory organs, and which appear due to decrease of red blood-corpuscle, by which the exchange of gases in the lungs is apparently effected. If the diaphragm be much pressed upward by the enlarged spleen, or if, as often happens, bronchial catarrh develop in the course of the disease, the dyspnœa may become very great. Such a combination of symptoms should always excite the suspicion that the patient is suffering from leuchæmia, and induce an examination of the blood. For this purpose we do not need a large venesection, and the debilitated and bloodless state of the patient almost always forbids this. In the blood that has been drawn we find at the border, between the buffy coat and the clot, single clumps, or a connected, loose, grayish layer, consisting of colorless blood-corpuscles. If, by beating, we free the blood that has been drawn of its fibrin, after standing for some time in a narrow glass, the heavy red corpuscles sink, and the lighter colorless ones form a whitish, purulent-looking, or milky layer in the upper part of the vessel. If we place a drop of the blood, recently drawn, under the microscope, we do not see a very few white corpuscles in the field, as we do in normal blood, but there are quantities of them which are not scattered around among the red corpuscles, but are more apt to be congregated in irregular clumps, as they are very adherent.

The course of the disease varies. In some, but, by no means, in all cases, besides the above symptoms, there is a hæmorrhagic diathesis. The patients have numerous hæmorrhages, from the nose particularly, more rarely from the intestinal canal, or into the tissue of the skin, sometimes into the brain. The fatal termination is hastened by this complication. The patients either die suddenly of apoplexy, or are so exhausted by repeated and abundant loss of blood that they soon die of exhaustion and anæmia. If a hæmorrhagic diathesis does not develop, the disease almost always runs a tedious course, and may even continue for years. In such cases the enlargement of the spleen and lymphatic glands reaches a very high grade; the tension of the capsule of the spleen, and the inflammatory irritation in it, developed, perhaps, by the tension, or by hæmorrhagic infarctions accompanying the hypertrophy, cause occasional pain in the region of the spleen and febrile symptoms. In these protracted cases the liver also is generally enlarged. The patients become much emaciated, and have a very pale, cachectic look; the dyspnœa increases, and becomes extreme. Sediments of the urates, or of pure uric acid, are very frequently found in the urine. It is possible that their formation is partly due to the dyspnœa and fever; but there is also a probability that the uric acid is formed by the higher oxidation of the hypoxanthin that is so abundant in the blood. In many cases there is bronchial catarrh, so that the patients have severe cough, with mucous expectoration. Still more frequently there is intestinal catarrh, which leads to obstinate diarrhœa. Dropsy often occurs toward the end. That this does not occur sooner, as we should expect from the analogy with other conditions where the patient is pale and cachectic, is doubtless due to the fact that, in leuchæmia, the decrease of the red blood-corpuscles is not accompanied by a corresponding decrease of the serum of the blood, as it is in other exhausting diseases. In the later stages of leuchæmia, the fever, which was at first temporary, usually becomes permanent. *Uhle*, who carefully measured the temperature, in one case, found a constant increase of one to one and one-half degrees during the latter weeks of life. If no complication occurs, death results from gradual exhaustion; it is often preceded by symptoms of disturbed brain-function, delirium, or stupor.

TREATMENT.—Up to the present time no case of recovery from leuchæmia is known, hence we cannot recommend any treatment that has actually proved successful. Quinine, iron, and iodine preparations have been used on account of their efficacy in some diseases of the spleen, and in anæmia. In the case of lymphatic leuchæmia that was under my observation, the enlargement of the glands temporarily subsided under the opposite mode of treatment, under the use of *Zittmann's* decoction. I afterward sent the patient to a water-cure establishment, where he improved and became healthy-looking. After a few months, however, the disease returned, advanced rapidly, and ended in death.

## CHAPTER II.

### MELANÆMIA.

ETIOLOGY.—In melanæmia there is found in the blood a granular pigment, partly free, partly enclosed in cells, partly embedded in small hyaline coagula. There is no doubt that this pigment comes from the coloring matter of the blood, but it is a question where and under what circumstances it is formed.

Almost all observers regard the spleen as the place where the pigment is formed in melanæmia. The frequent occurrence of pigmented cells in the spleens of animals, which some observers regard as physiological, others as pathological, as well as the fact that in melanæmia the pigment is almost always most abundant in the spleen, certainly favors the idea that the pigment is chiefly formed in the spleen, but does not prove that it is formed there only, and is not formed in other organs at the same time. *Frerichs* describes a case where he found no pigment in the spleen, while he found so much in the liver, that he was obliged to regard this organ as the place where it was formed.

The extensive occurrence of pigment in the blood presupposes extensive destruction of red blood-corpuscles. Whether this takes place exclusively in the spleen or whether it takes place in other organs at the same time, all observations prove that it is due to the influence of malarial infection. The milder forms of simple intermittent fever do not, however, appear to cause the formation of pigment in the blood at all or else only moderately, and only the severe and obstinate forms, but particularly the pernicious intermittent, appear to cause the higher grades of melanæmia in this country. The corresponding reports of physicians in the tropics, about the dark color of the different organs, particularly of the brain, in the bodies of patients that have had remittent fever, render it very probable that this form of malarial disease also constantly, or at least very frequently, causes melanæmia.

It is very probable that the dilatation of the blood-vessels and the consequent retardation of the current of blood (see page 711) become so great in pernicious intermittent, and in remittent marsh-fevers of the tropics, that the blood stagnates in the spleen. We might further suppose that the corpuscles in the stagnating blood are destroyed, and hence an altered pigment is developed from their hematin, processes which we often observe in stagnating extravasated blood. This explanation of the formation of pigment in a purely mechanical way is refuted by the fact that in intermittent fever the enlargement of the spleen, and consequently the retardation of the current of blood through it, may be very decided without the occurrence of melanæmia; and, on the contrary, melanæmia is found in cases where the spleen is only moderately enlarged. Hence we must suppose that marsh miasm has a pernicious influence on the red corpuscles in some other way that we do not yet know; and that in our country only in certain epidemics, but in the tropics in the endemic fevers, this influence frequently or constantly causes an extensive necrosis of the red blood-corpuscles, and the formation of pigment from its hematin (*Griesinger*).

*Virchow's* labors on the subject of pathological pigments readily explain why the pigment found in the blood appears not only as free granules, but also as enclosed in colorless cells. This observer saw that, in dissolving the hematin in a drop of blood by adding water, the hematin became most distinct in the colorless blood-corpuscles, and hence it is probable that, in the extensive destruction of blood-cells in the spleen, the hematin enters the colorless elements of the spleen-pulp, and with these reaches the blood. It is more difficult to explain the occurrence of pigment in the blood in the form of irregular flakes. It is possible that these flakes consist of fibrin that has precipitated on the angular granules; but it is more probable that the substance adherent to the pigment-granules, surrounding them like a bright border, consists of the protein substance that was combined with the fibrin in the blood-corpuscles that were destroyed (*Virchow*).

Anatomical Appearances.—In melanæmia the pigment found in the blood of the heart and vessels is black; more rarely besides the black we find brown or yellowish-brown, rarely yellowish-red pigment. With acids and caustic alkalies it shows the following conditions, which *Virchow* has found peculiarly characteristic of pathological pigment: the more recent formations become pale and finally lose their color entirely, while the older ones resist the reaction of these reagents a long time (*Frerichs*). The small pigment-granules have an irregularly roundish form. As *Meckel*, the first observer of pigment in the blood, saw, a larger or smaller number of these is almost always united, by a colorless substance, to roundish, spindle-shaped, or irregular flakes. The cells containing pigment sometimes have the size and form of the white corpuscles of the blood; sometimes they are larger, and club or or spindle shaped; the latter resemble the spindle-shaped cells in the spleen-pulp, which *Kölliker* considers the epithelium of the splenic vein. Besides these forms, *Frerichs* observed large clumps of pigment of irregular shape, as well as cylindrical bodies that looked like small vessels.

With the blood the pigment enters all the organs of the body, and, according to the amount collecting in the capillaries, colors them more or less. According to *Planer* and *Frerichs*, we almost always find the most pigment in the spleen, so that it appears slate-gray and often almost black. Next to the spleen, the greatest amount of pigment is found in the liver and brain, particularly in the cortical substance. The liver is often steel-gray or blackish; the cortical substance of the brain chocolate or graphite color. Not unfrequently there is also a considerable collection of pigment in the kidneys, as a result of which usually the cortical substance has gray points in it. In the pulmonary vessels, particularly in the smaller ones, there is occasionally a large amount of pigment. In the vessels of the other tissues and organs it is never accumulated to any great extent; but the skin, mucous membranes, cellular tissue, and lymphatic glands, also have a more or less decidedly gray color. *Frerichs* sums up the appearances in melanæmia by saying: "In marked cases of the disease pigment is found wherever the blood goes, and is the more abundant the smaller the capillaries of the part, that is, the more readily impaction of the flakes may occur."

SYMPTOMS AND COURSE.—Many cases of melanæmia cause no perceptible disturbance of function, and the organs have been found overloaded with pigment, on autopsy of persons who have died of the most varied diseases. This was true in more than one-third of the cases observed by *Planer*. On the other hand, patients often die quickly with severe brain-symptoms, and on autopsy we find the evidences of melanæmia, particularly an accumulation of pigment in the vessels of the brain, or numerous small extravasations of blood through the brain-substance. Former observations of great pigmentation of the cerebral substance in persons who had died of comatose intermittent, as well as the experience of the tropics, which shows that the brain is very dark-colored in most persons who have died of severe remittent fever, have acquired greater significance, since we know that the dark color of the brain depends on an accumulation of pigment in its vessels, and have rendered it very probable that the obstruction of the cerebral vessels (with or without consecutive rupture of the walls of the capillaries) caused the brain-symptoms in severe malarial diseases. This view, advanced by *Meckel*, appeared to be supported by numerous observations of *Planer* and *Frerichs*. Both of these observers described cases of melanæmia where the patients had severe cerebral symptoms, either headache and dizziness, delirium or convulsions, but particularly coma. The cases observed by *Frerichs* all occurred during a malignant epidemic of intermittent fever; they developed partly from simple intermittent paroxysms, had an irregular intermittent or remittent course, and partly yielded to quinine; they presented exactly the symptoms of a febris intermittens comitata, maniaca, epileptica, comitosa, apoplectica. With all this there are important reasons for doubting the dependence, at least the constant dependence, of the cerebral symptoms in pernicious malarial fever on an obstruction of the vessels of the brain by pigment. In opposition to the hypothesis of such a connection, *Frerichs* calls attention to the fact that in many cases, even when the brain is very dark-colored, no decided disturbance of the circulation can be perceived; and further, that in spite of the dark color of the brain, there are often no cerebral symptoms; and lastly, that severe cerebral symptoms have been observed where there was none of this pigmentation of the brain (in twenty-eight cases of intermittens cephalica that *Frerichs* observed, the dark color of the brain was absent in six). The typical occurrence of these brain-symptoms, as well as the occasionally successful treatment of them by quinine, appears to me just as weighty an argument against their dependence on obstruction of the cerebral vessels by pigment. For it is impossible to see how the obstruction should exist during the paroxysm and disappear during the apyrexia, and there is just as little probability that the exhibition of quinine could have a favorable effect on obstruction of the cerebral vessels.

According to what has been said, with our present knowledge, we can affirm nothing certainly of the connection between melanæmia and the disturbance of the brain function. It is certainly possible that the poisoning of the blood by marsh miasm, in malignant intermittent fever, may excite other cerebral disturbances *besides* accumulation of pigment in the cerebral vessels, and independently of that.

In another series of cases, *Planer* and *Frerichs* found anomalies of the renal functions in melanæmia. Occasionally the secretion of urine was entirely arrested; in other cases there was albuminuria; in still others, hæmaturia. The same objections urged against the dependence of the brain-symptoms on obstruction of the cerebral vessels must be equally valid against the dependence, at least the constant dependence, of functional disturbance of the kidneys on obstruction of the renal vessels. Among others, *Frerichs* saw albuminuria occur without pigmentation of the kidneys; and, on the other hand, albuminuria was absent in five cases where there was pigment in the kidneys. But, if there was albuminuria, this observer found that the amount of albumen in the urine was decidedly increased during the paroxysm, and decreased or disappeared during the interval. From analogy with other miasmatic affections, it certainly cannot be denied that the infection of the blood with marsh miasm may disturb the nutrition and functions of the kidneys, even without obstruction of the vessels.

Lastly, in melanæmia *Frerichs* observed exhausting intestinal hæmorrhage, profuse diarrhœa, acute serous effusions into the peritoneal sac, and bloody suffusion of the serous coat of the intestines. It appears improper to refer this symptom also to obstruction of the vessels of the liver, and to congestion in the roots of the portal veins; for, although in all the cases observed by *Frerichs*, next to the spleen, the liver appeared to contain the most pigment, the symptoms that apparently indicated disturbance of the circulation in the roots of the portal veins were not by any means constant, and were not nearly so frequent as the cerebral symptoms. Moreover, the intestinal hæmorrhages observed by *Frerichs* in three cases had distinct intermissions; and, while they resisted treatment directed against the hæmorrhage directly, they yielded to large doses of quinine. *Frerichs* even refers the fatal result in one of these three cases to neglect in the administration of quinine. In these cases, also, it would be difficult to believe in an intermittent obstruction of the blood-vessels.

It may be offered as an objection to referring the above symptoms to melanæmia, that there are but few symptoms that we certainly know belong to that disease itself, and are not the immediate effects of malarial poisoning. Among these are the dark color of the skin, which is caused by the quantity of pigment in the blood-vessels of the skin, and the microscopical observation of pigment in the blood. In mild cases the skin is of an ashy-gray color, in severe ones it is yellowish brown. If we find this color in a person who has had an obstinate and severe intermittent fever for some time, or if it appears that this intermittent belonged to a malignant epidemic, and had the symptoms of a febris comitata, we should strongly suspect melanæmia, and make a microscopical examination of the blood.

TREATMENT.—Prophylaxis and the causal indications demand the same general rules as we shall give, in the second volume, for the treatment of pernicious intermittent fever. We cannot fulfil the indication from the disease, as we know no remedy that will remove the pigment from the blood. In recent cases the symptomatic indications may require the administration of iron, and a nourishing diet; for, as *Frerichs* justly says, the excessive loss of red corpuscles causes chlorosis, as well as melanæmia. Later the former often disappears, while the latter continues. In one case that I saw, several months passed before the patient recovered under the use of iron; subsequently he was equal to the hardest work, and presented no anomaly except the color of his skin. When he died, several years afterward, of pneumonia, the autopsy still showed distinctly the signs of melanæmia.

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